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PREFATORY NOTE

THE FOUR PAPERS included in this number were in preparation for publication at the time of Dr. Dusser de Barenne's death on June 9, 1940. His colleagues, Drs. McCulloch and Garol, who were collaborating with him in his investigations, have completed the work and prepared the papers for press. It seemed to the Editors of the *Journal* that the papers, if published together, would constitute a fitting tribute to the memory of their former colleague. The papers are therefore being issued together in this special number which has been financed by private subscription.

A bibliography of Dr. Dusser de Barenne's scientific writings was appended to the appreciation which appeared in the *Journal* for July 1940 (pp. 283–292). To complete this list we may now add the following titles which have appeared since that time:

DUSSER de BARENNE, J. G., GAROL, H. W. and MCCULLOCH, W. S. Sensory cortex of chimpanzee. *J. Neurophysiol.*, 1940, *3:* 469–485.

MURPHY, J. P. and DUSSER de BARENNE, J. G. Thermocoagulation of motor cortex exclusive of its sixth layer. *J. Neurophysiol.*, 1941, *4:* 147–152.

DUSSER de BARENNE, J. G., MARSHALL, C., NIMS, L. F. and STONE, W. E. The response of the cerebral cortex to local application of strychnine nitrate. *Amer. J. Physiol.*, 1941, *132:* 776–780.

DUSSER de BARENNE, J. G., GAROL, H. W., and MCCULLOCH, W. S. The "motor" cortex of the chimpanzee. *J. Neurophysiol.*, 1941, *4:* 287–303.

DUSSER de BARENNE, J. G., and MCCULLOCH, W. S. Functional interdependence of sensory cortex and thalamus. *J. Neurophysiol.*, 1941, *4:* 304–310.

DUSSER de BARENNE, J. G., and MCCULLOCH, W. S. Suppression of motor response obtained from area 4 by stimulation of area 4s. *J. Neurophysiol.*, 1941, *4:* 311–323.

DUSSER de BARENNE, J. G., GAROL, H. W., and MCCULLOCH, W. S. Functional organization of sensory and adjacent cortex. *J. Neurophysiol.*, 1941, *4:* 324–330.

In press

DUSSER de BARENNE, J. G. A method for uniform sectioning of brain, *J. Tech. Methods and Bull. internat. Ass. med. Museums.*

DUSSER de BARENNE, J. G., GAROL, H. W., and MCCULLOCH, W. S. Physiological neuronography of the cortico-striatal connections. *Res. Publ. Ass. nerv. ment. Dis.*

THE EDITORS

THE "MOTOR" CORTEX OF THE CHIMPANZEE*

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(Received for publication April 22, 1941)

INTRODUCTION

IN PREVIOUS papers dealing with the sensory cortex of the chimpanzee[1,2,29,37] we have mentioned that in the animals upon which those experiments were performed an exploration of motor response to electrical stimulation of the cerebral cortex preceded each investigation of the location, subdivision or functional organization of the sensory cortex. These explorations differ in four respects from those reported by other investigators.[4,54,55,56,59,61] (i) The subsequent studies on the sensory cortex necessitated the use of a head holder which so fixed the jaws and immobilized the head that the tongue could never be observed and contractions of neck musculature were manifest only to palpation. (ii) The chimpanzees were under Dial narcosis of such a depth that there was usually much tension of the extensor and frequently moderate tension of the flexor muscles. (iii) The carefully controlled electrical stimulation was usually by very long pulses, often at extremely low frequencies. (iv) The area stimulated was not confined to the precentral convolution but extended over the entire exposed hemisphere, *i.e.*, from in front of the "motor" eye field to behind the sulcus lunatus and from as far medial as could be reached with the stimulating electrodes, without displacing the brain, to the fissura Sylvii laterally. In this paper are recorded all phenomena obtained by stimulation anywhere in this extensive area.

METHODS

Nine chimpanzees (*Pan satyrus*) 2½ to 3½ years old were used altogether. The animals were given 0.35 to 0.45 cc. Dial§ per kg. body weight, ½ intraperitoneally, ½ intramuscularly. This produced full anaesthesia without abolishing muscular tension. The narcotized animal was placed on a board with the hind quarters elevated to maintain good cerebral circulation and with the head fixed to the board by one bar over and behind the lower incisors and a second behind the occiput.

One hemisphere was then exposed by turning down a large osteoplastic flap. In opening the dura mater special care was taken not to injure the cerebral veins passing to the dural lacunae. Photographs from several angles were taken and printed and the location of each stimulation was recorded on these as well as on drawings of twice life-size.

In most of the experiments the contralateral arm and leg were then suspended by heavy rubber bands so disposed as to allow freedom of movement without compromising circulation. In these cases the motions were noted by two or more observers and any change in muscular tension was controlled by palpation and passive movement of the part affected.

In the earliest of these experiments an ordinary thyratron stimulator (after Schmitt[74]) was employed. Thanks to Craig Goodwin, electronic engineer for this laboratory, it was later replaced by an instrument of greater flexibility.[52] This permits independent control

* Aided by a grant from the John and Mary R. Markle Foundation.
† Deceased June 9, 1940.
‡ National Research Fellow 1940–1941.
§ We wish to thank the Ciba Co. for kindly putting the Dial at our disposal.

of voltage, of number of pulses per second, of wave form, the ascending and descending phases being separately controlled, and finally, delivery of a train of impulses of any required length. When, with this new apparatus, it had been found that, except in cases of excessive narcosis or impaired cerebral blood supply, a very short ascending phase with a falling phase of from 5σ to 20σ was effective with lower voltage than sufficed with other wave forms giving comparable extinction, a simpler thyratron stimulator was constructed by Mr. Goodwin and has been employed ever since for mapping the "electrically excitable" cortex (see Fig. 1). It gives pulses at frequencies of 1 per sec. to 100 per sec. with a short ascending phase and a descending phase which can be varied from 0.5σ to 20σ. This device, like its predecessor, has an output stage designed to give practically constant voltage although the circuit through the cortex varies from 500 to many thousand ohms. With this output equal responses can be elicited at relatively constant voltage despite gross differences in the area of contact between electrodes and brain, an important consideration when so-called stigmatic electrodes are manually applied.

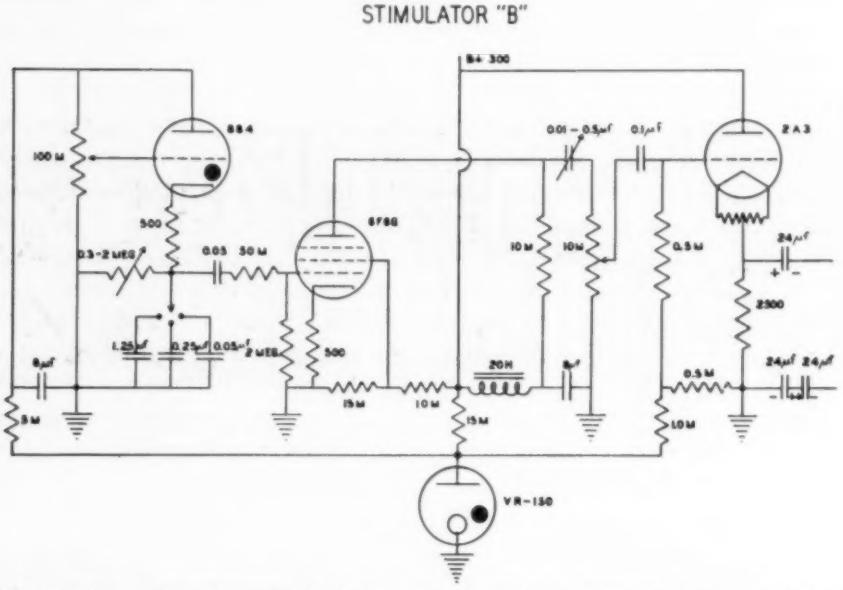


FIG. 1. Circuit of stimulator "B" which gives a variable falling phase of 0.5 to 20.0σ and a frequency of 1 to 100 per second. (Designed by Craig W. Goodwin)

Stigmatic Ag-AgCl electrodes, bipolar and monopolar, were used and gave essentially similar results, except that the bipolar method with a relatively dry brain produced its motor responses at a lower voltage. Since physical considerations indicate that the bipolar method is less likely to stimulate underlying fiber tracts, quite apart from the lower voltage required, this method was generally preferred. As the voltage required for stimulation decreased until the electrodes were about 3 mm. apart and as very little change was noted when they were separated further, this distance was the one generally employed.

The frequencies used varied from 1 to 40 per sec. In general, the greater the frequency, the shorter was the duration required to elicit a motor response. In no case was stimulation prolonged enough to produce extinction,[26–28,31–33,35,63] except in one experiment designed to investigate it. As a further precaution against confusing results by either extinction or facilitation, a minute or more was allowed to elapse between successive stimulations and, even then, the second stimulation was not applied in the region of the first. For experiments in which facilitation[6–10,12–15,35,42,62] was desired, frequencies of about 30 per sec. were employed and the electrodes moved rapidly from point to point.[11] For the analysis of facilitation and extinction the wrist was fixed and the movements of the finger recorded isotonically on smoked paper and a focal cortical point for flexion of this digit was stimulated.

Although in seven of the chimpanzees both hemispheres were stimulated most of the findings reported here were obtained from the first hemisphere, for, while the second was usually in good condition when exposed, the animal had then been on the table for a matter of a day or more, the threshold had gone up and responses were less discrete. Thus only a rough map of it was warranted.

Findings

The time relations for primary facilitation (*i.e.*, the fall in threshold, diminution of latency or increase in amplitude induced by antecedent stimulation of one and the same focus) and extinction (*i.e.*, the rise in threshold, increase in latency or decrease in amplitude induced by antecedent stimulation of one and the same focus) and their dependence upon the parameters of stimulation are grossly similar to those found in the macaque (see Fig. 2a). The response, however, is more complicated in character and at values of stimulation near threshold the latency is much less.

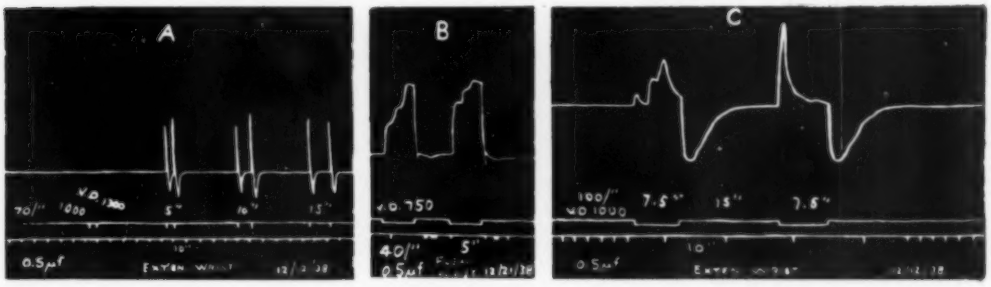
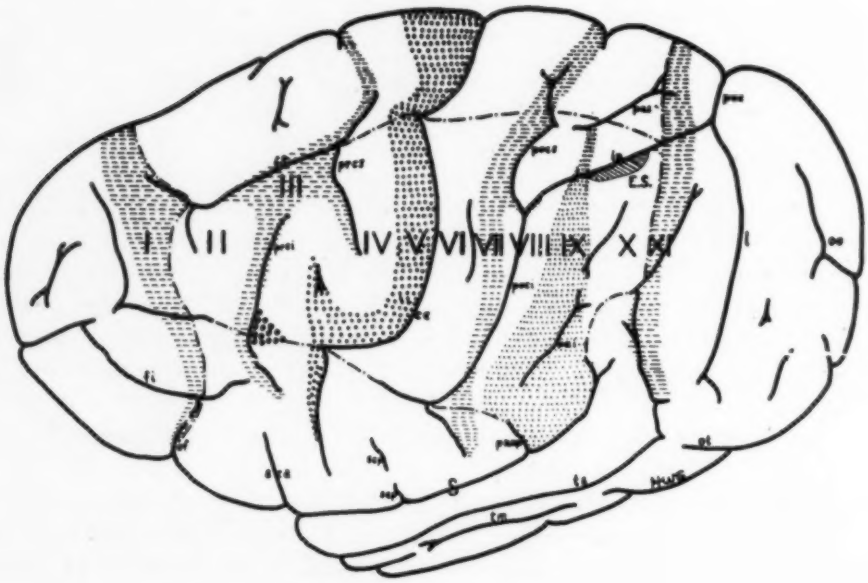


Fig. 2. Isotonic recording via air tambours. Monopolar stimulation. 0.5μf. A. Shows facilitation with intervals of 10 sec.; with partial extinction at 15 seconds. B. Shows triphasic response with more marked facilitation of the earlier components. C. Triphasic response showing facilitation of first component with extinction of subsequent components. In each case the response is followed by a contraction of the antagonistic muscles.

If the stimulation is continued for 2 to 2.5 seconds the response usually consists of 3 components. The first of these occurs, even at threshold values, with a latency too small to detect on the kymographic record. The second component appears at least a second later and the third about a second later still. With weak stimulation the second is larger than the first and the third is larger than the second. With stronger stimulation the first is disproportionately larger. No fourth component appears even when stimulation is prolonged to 7.5 sec. (see Fig. 2b). With respect to facilitation and extinction the components behave differently. As extinction is enhanced (by increasing the frequency or the duration of stimulation or by increasing the interval) the later components, particularly the third, diminish or disappear, while the first still exhibits facilitation (see Fig. 2c).

Motor bands—The lowest threshold was found immediately anterior to the fissura centralis, *i.e.*, Band V (see Fig. 3). Immediately frontal to this band, *i.e.*, in Band IV, the threshold was definitely higher. (Both of these regions gave highly specific movements.) Even in these regions the thres-



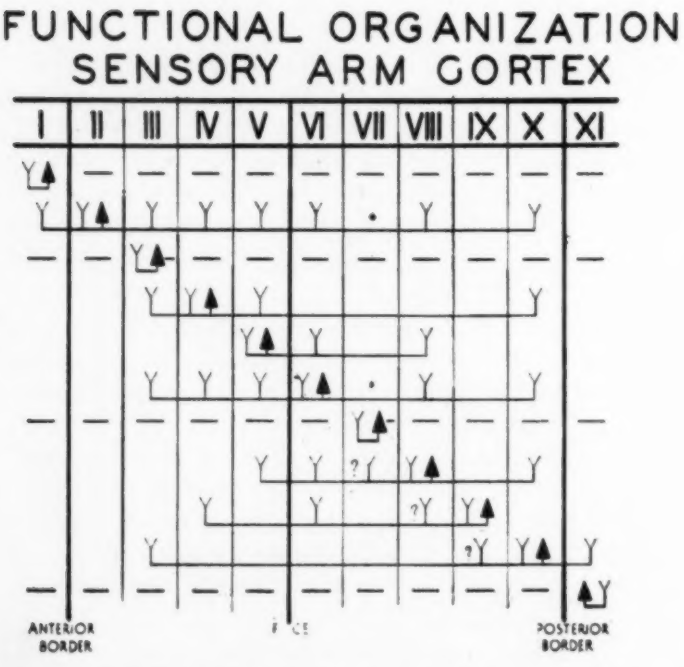


FIG. 3. *Above.* The extent, location and functional subdivisions of the sensory arm cortex into physiologically distinguishable bands, No. II–X, and of the immediately adjacent bands, No. I and XI, indicated on a composite drawing representing the arm area in the center of the field. The bands giving suppression, No. I, III, VII and XI, are marked thus: ≡≡≡≡. Small ⁞⁞⁞⁞ indicate Bands V and IX. Large ⁞⁞⁞⁞ mark the "dud" areas. Areas between trunk and arm and between arm and neck are marked—·—·—·. (Divisions between neck and face, and trunk and leg not marked.)\\\\\\\\ mark the visuo-

hold was higher in the sectors for neck and truck than in those for face, arm and leg.

Band II gave mixed motor responses of leg and arm, of arm, eyes and face, and even of leg, arm and face in its most antero-medial portion. When the animal was lightly narcotized and in good condition these responses were readily elicited, whereas when it was deeply narcotized they could be obtained only after facilitation or prolonged stimulation. In the latter case they were frequently followed by motor after-discharge.

Approximately the anterior half of the postcentral convolution, Band VI, gave discrete movements which were different from those elicited from foci immediately precentral to them and that without altering the stimulus. This presumably eliminates spread of current as the efficient cause of these responses.

T. Graham Brown defines secondary facilitation: "The facilitation of a cortical point therefore seems to raise the excitability of a wide area of the surrounding cortex for the reaction evoked by the stimulation of that point."[11 (see p. 122), 1.20] By secondary facilitation, starting from either the pre- or postcentral gyrus, movements were obtained from Bands VIII and IX providing there was appreciable muscular tension in the extremity involved. This was repeatedly done only in the arm region where our investigations were most extensive.

Suppressor bands[30,34,39,51,57]—Stimulation of Band I, III, VII or XI, even with 3 or 4 times the voltage which elicited response from adjacent motor foci, failed to elicit movement. Yet it had the following effects.

First, it suppressed motor response to electrical stimulation,[36,38] *i.e.*, after stimulation of any of these bands stimulation of a motor focus with a stimulus which previously elicited a motor response now failed to do so.

Second, it suppressed motor after-discharge following cortical stimulation, *i.e.*, during several seconds of stimulation of any one of these bands the clonic contractions initiated by stimulation of Band II, IV, V or VI were held in abeyance. This could be repeated several times during a single motor after-discharge. More prolonged stimulation of these bands terminated it and prevented its reappearance.

Third, it relaxed existing muscular contraction, *i.e.*, stimulation of any of these bands caused a disappearance of resistance to passive motion, a softening of muscle bellies as judged by palpation, and a falling in response to gravity of an extremity previously supported only by its muscular tension.

In order to make clear the nature of these explorations and the type of

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sensory band of Elliot Smith. *Below.* Diagrammatical representation of the directed functional (and anatomical) relations between the various cortical bands of the arm-subdivision of the sensory cortex found in these experiments and also those of Bands I and XI adjacent to but outside of the sensory cortex. Anterior and posterior borders are the limits of the sensory cortex. The suppression of the ECG of various bands upon strychninization of Bands I, III, VII and XI is indicated thus: ————. F CE = fissura centralis; * = no certain evidence; ?Y = definite "firing" but uncertainty whether strychnine invaded region so "fired."

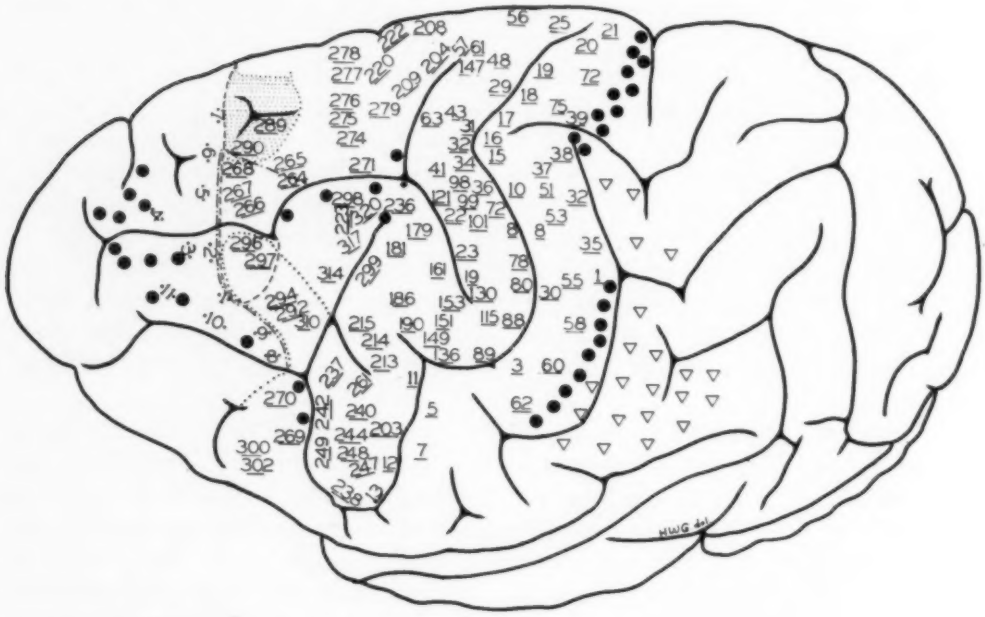

FIG. 4. Chimpanzee 11. May 28, 1940.

● Electrical stimulation here elicited
  a) relaxation of existing muscular tension
  b) suppression of motor response
  c) holding in abeyance motor after-discharge

▽ Secondary facilitation elicited motor response only when there was existing muscular "tension" in contralateral limbs.

⁙ Stimulation here elicited simultaneous progressive movements[15,17] of both fore and hind limbs, most marked on the contralateral side, but also some ipsilateral,[17] accompanied by alternating forceful expirations and occasionally with suggested vocalization.

– – – The anterior boundary of the area eliciting motor response of the hind limb, upon facilitation.

–·–·– The anterior boundary of the area eliciting motor response of the fore limb, upon facilitation.

•••• The anterior boundary of the area eliciting motor response of the face, upon facilitation.

✱ Motor point elicited contraction of the latissimus dorsi contralaterally.

The numbered points indicate sites from which *primary responses* were obtained. They are divided into three groups: (a) precentral, (b) postcentral and (c) eyefield. The latter points are indicated by two dots adjacent to the numbers, corresponding to the exact site and separation of electrodes. The numbers of the other two groups are underlined, indicating the site of the electrodes, their distance apart being uniformly 3 mm.

a) Precentral motor points

V.D. 3000, f.p. 10σ, 40 per sec., bipolar

8 Flexion of elbow
11 Lower lip to right
12 Entire mouth to right
13 Entire mouth to right
19 Pronation of wrist
22 Internal rotation of shoulder
23 Flexion of elbow

V.D. 1500, f.p. 10σ, 5 per sec., bipolar

25 Plantar flexion
29 Flexion of hip
31 Adduction of thigh with slight flexion of hip
32 Contraction right rectus abdominis
34 Contraction in region of serratus anterior
36 Adduction of shoulder
41 Contraction of serratus anterior
43 Adduction of thigh
48 Plantar flexion

Description of Fig. 4—Continued

V.D. 1500, f.p. 10σ, 1 per sec., bipolar
56 Opposition of hallux
V.D. 1500, f.p. 10σ, 50 per sec., bipolar
57 Flexion of all toes with inversion of foot
61 Flexion of toes with inversion of foot
63 Extension of knee
V.D. 1500, f.p. 10σ, 1 per sec., bipolar
72 Flexion of elbow—mainly m. brachialis
78 Flexion of elbow—mainly m. biceps
80 Flexion of elbow with slight pronation
V.D. 1500, f.p. 10σ, 50 per sec., bipolar
88 Flexion digits 2, 3, 4 and 5 of hand
89 Flexion digitis indicis only
V.D. 1500, f.p. 10σ, 1 per sec., bipolar
98 Contraction of trapezius
99 Contraction of anterior half of m. deltoideus
101 Flexion of elbow—m. biceps alone
115 Adduction digiti minimi quinti of hand
121 Contraction in posterior axillary fold (latissimus dorsi)
130 Pronation at wrist (pronator teres and ? pronator quadratus)
136 Flexion digiti indicis only
147 Dorsiflexion foot
149 Flexion digiti indicis only
151 Flexion digiti minimi quinti only
153 Flexor carpi radialis
161 Slight contraction triceps
179 Contraction triceps only
181 Flexor carpi radialis
186 Flexor digitorum sublimis
190 Flexor digitorum profundus
203 Retraction corner of mouth
204 Plantar flexion
208 Plantar flexion
209 Plantar flexion
213 Elevation of lower lip
214 Drew down and protruded lower lip
215 Right orbicularis oculi with retraction of pinna
220 Dorsiflexion of foot
222 Plantar flexion
223 Flexion elbow (biceps)
V.D. 2500, f.p. 20σ, 1 per sec., bipolar
236 Right rhomboids only
237 Lower lip to right
238 Mouth to right
240 Mouth to right—chiefly lower lip
242 Both lips to right
244 Lower lip to right
247 Lower lip to right
248 Lower lip to right
249 Lower lip with slight upper lip to right
261 Mouth to right, chiefly lower lip
264 Flexion in flank with slight flexion of hip
265 Flexion of hip with slight flexion of flank
266 Flexion of hip
267 Flexion of knee
268 Flexion of knee
269 Lower lip to right
270 Lower lip to *left* with protrusion of lips
271 Latissimus dorsi—adduction and retraction of arm
274 Deep flexors of hip (iliopsoas?)
275 Flexion of knee
276 Dorsiflexion foot
277 Dorsiflexion foot
278 Plantar flexion foot
279 Plantar flexion foot
289 Flexion of pelvis, hip and knee
290 Contraction in flank
292 Pronation of forearm
294 Flexion of fingers (flex. dig. profundus)
296 Supination (brachioradialis)
297 Flexion of fingers (flex. dig. profundus)
298 Flexion elbow (biceps with brachioradialis)
299 Flexion and pronation of wrist. Right orbicularis oculi only (contralateral blinking) obtained from here to just rostral of 237
300 Mouth to right
302 Mouth to right
310 Pronation of forearm: later slight flexion of fingers
314 Pronation of forearm (pronator teres)
317 Slight flexion of forearm (brachialis)
320 Slight flexion of forearm

b) Postcentral motor points
V.D. 2500, f.p. 20σ, 7 per sec., bipolar
1 Pronation forearm
3 Flexion of fingers (flex. dig. profundus)
5 Lower lip to right
7 Corner of lower lip drawn down
8 Extension of elbow internal rotation of shoulder
10 Latissimus dorsi—adduction of shoulder
15 Contraction of abdominal wall

Description of Fig. 4—Continued

16 Flexion at hip (iliopsoas?)
17 Flexion of hip
18 Extension of knee
19 Plantar flexion of foot
20 Opposition of hallux
21 Flexion of all toes
30 Pronation forearm with slight flexion at elbow
32 Internal rotation of shoulder with adduction of arm
35 Pronation forearm
37 Deep muscles of shoulder girdle
38 Trunk muscles (erector spinae?)
39 Flexion of hip
51 Adduction of shoulder
53 Flexion of elbow (biceps)
55 Flexion of elbow (biceps) with pronation
58 Flexion all digits of hand
60 Flexion all digits of hand
62 Flexion of index finger only
72 Dorsiflexion of foot
75 Extension of knee

c) Eyefields
V.D. 1500, f.p. 10σ, 45 per sec., bipolar
1 Oculi contra and slight upward rotation
2 Oculi contra only
3 Oculi contra only (same with V.D. *200*)
4 Same
5 Same
6 Same but less rapidly
7 Oculi contra but slowly
8 Oculi contra
9 Same
10 Same
11 Same

evidence summarized in the foregoing statements of the findings, we would cite a fair sample of the results of stimulation of the left hemisphere of chimpanzee 11, on which over 500 individual stimulations yielded discrete responses. These are given in Fig. 4 and its legend.

Discussion

A survey of previous results obtained by electrical stimulation of the cortex in any species[4,13,16–18,22,43–48,54–56,59,61,65,67,68,70–73,77,80] discloses great variation in findings, not only from observer to observer but from observation to observation by the same observer. The work on the chimpanzee is no exception.

When one seeks the reasons for the variation he finds these fall into one of three groups related either to the animal used, the conditions of the experiment or the type of stimulation.

Most, if not all, of the electrical stimulations reported on the chimpanzee[8,13,54–56,61] have been on *Pan satyrus* but the age and physical status of the animals have varied widely. As the group reported here ranged from 2.5 to 3.5 years and were all in good nutritional state they were to this extent alike. The configurations of the hemispheres on the other hand were so variable, even between the two hemispheres of the same animal, as to render a comparison of detailed results difficult. Fortunately, the mapping of the functional organization of the sensory cortex, which followed each stimulatory investigation, permits one to be sure in which physiologically unique band a given stimulation was performed. Without this information no such general map as Fig. 5 could be justified. Even then, however, the difficulties of fitting the findings on any particular hemisphere into the schema representing all hemispheres (those in our collection as well as those described by Mingazzini,[66] Retzius,[69] Connolly[21] and others) must be kept in mind.

Most of the electrical stimulations of the cortex of the chimpanzee have been done under ether or ether and chloroform anaesthesia. In the group re-

ported here ether was administered only long enough to render the animal manageable for administering Dial. Under its influence the threshold for electrical stimulation of the cortex remains low and relatively constant in contrast to the findings of Bucy[16,17] and Bucy and Fulton.[18] Moreover, operations performed under Dial are not attended by vasodilatation or a rise in blood pressure commonly encountered when ether is used. While there is no generally accepted method for measuring the depth of narcosis, it may be appropriate to state that, under Dial as employed here, the electrocorticogram

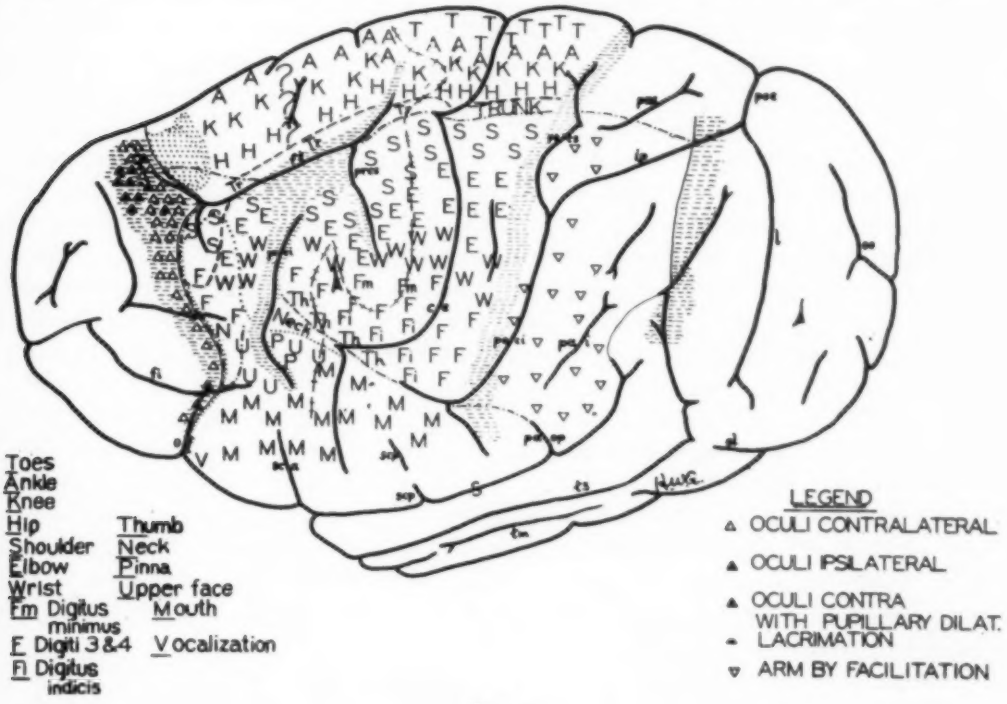

FIG. 5.

was essentially similar to the electroencephalogram found in man in normal sleep,[23] but the tension of the muscles was relatively high, holding the limbs in a persistent state of semiflexion, although the corneal reflexes were absent and no responses could be elicited by nocuous stimuli.

The exposure of the brain was made with extreme care, without trauma to the brain or to the dural vessels draining it except those which had to be thermocoagulated to permit a proper field of experimentation. In all cases enough vessels remained to ensure adequate circulation. The arachnoidal membrane over the cerebral hemisphere was not ruptured and the air of the room was kept around 80°C. and of high humidity, but the surface of the arachnoid membrane was not irrigated with saline solution as is done during operations on the human brain. The cerebrospinal fluid in the subarachnoid spaces was relied upon to keep the underlying cortex moist. Neither inspissation nor any unexpected threshold changes[20,53] were encountered in the

first ten hours. Thereafter inspissation began, threshold rose and pledgets of warm Ringer's solution had to be applied at intervals in order to restore electrical excitability to about its previous level.

The fall in blood pressure under Dial[50] was compensated by elevating the foot of the animal board so that the heart was on the same level as the head.

Ag-AgCl electrodes, small spherules on the end of long pliable silver wires, were applied lightly to the cortex and that only during stimulation, thus minimizing local ischemic changes.

Type of stimulation—The first group of experiments reported here, namely those of facilitation and extinction, are sufficient to indicate how the responses obtained depend upon the parameters of stimulation. As has been indicated in previous studies on the macaque,[35] it is indeed easy to alter and even reverse the response[13] elicited from a given focus by antecedent stimulation there or elsewhere, provided it be of the right kind and at the appropriate interval. This is, in fact, sufficient to account for such phenomena as deviation of response, reversal of response, etc. It was to prevent just these alterations induced by stimulation that the type of stimulation here employed was devised for mapping the cerebral cortex. Higher frequencies, longer pulses and greater duration of stimulation are too apt to induce extinction, which disappears only after a prolonged interval, thus delaying the experiment. The higher voltage required with shorter wave forms, shorter periods of stimulation and lower frequencies inevitably increases the spread of current, and thus induces relatively complicated or diffuse movements. If time were no object one would, of course, select approximately 60-cycle stimulation to obtain even more discrete results, whereas, if the object were to obtain the maximum number of responses in a given time, he would select very short, high voltage thyratron pulses and expect diffuse results, and even those from only a small fraction of the cortex which can yield motor response upon adequate stimulation.

While everything mentioned above doubtless plays some part in determining the differences in findings, probably the biggest factor responsible for the relatively small area which yielded motor responses to the earlier investigators was the unsuspected presence of suppressor bands.[2,34] Even when their existence is known, until they have been delimited they remain most disturbing. For when one seeks the anterior or posterior margin of a region yielding "motor" response and, by chance, stimulates a band before or behind that region he induces a suppression of motor response which, unless the muscular tension be sufficiently great for relaxation to appear as a response, is manifest only as an inability to obtain a motor response from a point which previously yielded one to the same stimulus. The works of Leyton and Sherrington and of Graham Brown suggest that accidental stimulation or spread of current to Band III prevented them from obtaining responses from the more anterior portion of Bands IV and V, and, similarly, that accidental stimulation of Band VII prevented responses from Band VI, though this may have depended partly on the wave form which they used. It is

doubtful whether the experiments presented here would have been any more successful, had we not been forewarned by the results of our previous work on the suppression of motor response in the macaque.[36,38]

That there was a significant spread of current in the experiments of Leyton and Sherrington is suggested from the size and shape of the motor eyefields, as shown in their diagram (Fig. 6). As the length of the stimulating impulse is increased and the voltage diminished, the eyefield becomes narrower.

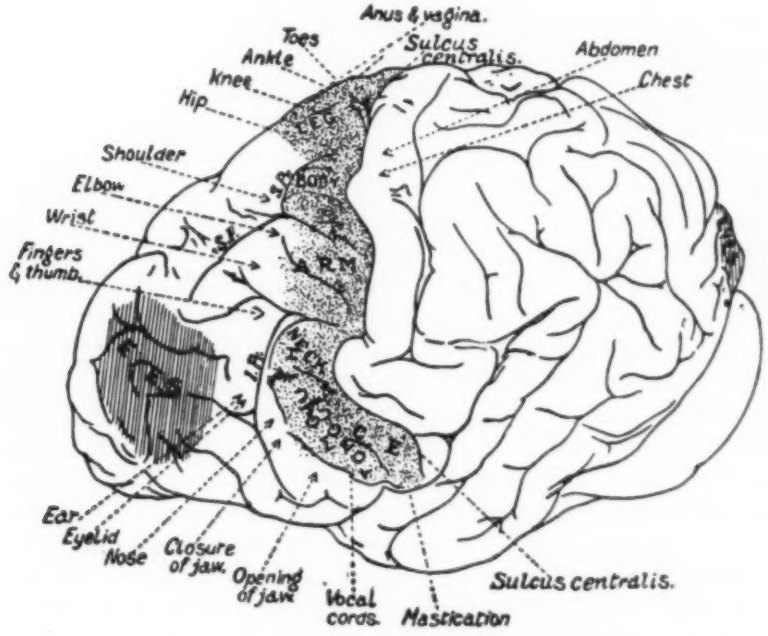

FIG. 6. From Grünbaum and Sherrington.

For example, when one traces point to point by "secondary facilitation" from the sulcus centralis, anteriorly or posteriorly, the suppressor bands interfere with the procedure, for if, in so doing, these bands are stimulated, no further responses are obtainable. One has, therefore, first to delimit these suppressor bands so that he can omit stimulation within them and resume it beyond them. A typical example for the anterior boundaries of leg, arm and face area is given in Fig. 4, as is also an indication of the far postcentral regions from which one can obtain motor responses by secondary facilitation, starting from foci in the postcentral gyrus which yield primary motor responses. It should be noted, however, that the response elicited from these far postcentral regions depends on the presence of muscular tension in the animal, a condition which is prevented by stimulation of Band VII or any other suppressor band. This is of especial interest for, while the nature and distribution of the factors for facilitation and extinction are fairly well known, neither the mode nor the site of operation of suppression of motor responses is yet established. Yet it would be as premature to attempt to con-

jecture what part this loss of tension may play in suppression of motor response as it would to attempt to explain how stimulation of the suppressor bands can hold in abeyance motor after-discharge following cortical stimulation. The outstanding fact which will have to be taken into consideration, in forming any hypothesis as to suppression of motor response, is this: it is obtainable from exactly the same areas as those which yield a suppression of electrical activity of the cerebral cortex.[2]

A comparison of the sensory cortex of the chimpanzee (Fig. 5) with that of the macaque[30] (see fig. 1) reveals an essential similarity of functional organization which not even the great discrepancy in size, shape and functional differentiation of the hemisphere can conceal.

It was hoped that in the work on the chimpanzee finer differentiations of function could be distinguished, as, indeed, was sometimes possible. However, the results with respect to Band I were in the main disappointing, for, while it was possible to find separate foci for a large number of discrete eye movements, in every hemisphere these occurred among foci for suppression, whether judged by relaxation of muscular tension, suppression of motor response or holding in abeyance an after-discharge.

Band II, lying immediately behind Band I, shows strikingly similar properties in both its "motor" and its "sensory" functions. It will be recalled that strychninization of any subdivision of this, the most anterior band of the sensory cortex, produced disturbances which were not confined to the subdivision of the cortex strychninized and in this same band, though primary movements restricted to some part of the extremity subserved by the subdivision in question could be elicited, experiments involving facilitation showed a wide overlapping seen nowhere else in the sensory cortex. The anterior boundary of Band II is fixed and easily found because of the suppression elicited from the band immediately in front of it. The same is true of its posterior boundary throughout the arm and part of the face regions, but in the leg region the posterior boundary is more difficult to locate, lying anterior to the ascending ramus of the superior precentral sulcus.

If one regards the superior precentral and the adjacent superior frontal sulci as comparable to the superior precentral in the macaque, and the ascending ramus of the inferior precentral as a homologue of the spur of the arcuate, Band III obviously represents area 4s, the "strip" in the macaque.[57] This should indicate a cytoarchitectonic division between these areas which Brodmann numbered 4 and 6 and which have been called respectively the "motor" and "premotor" regions.[49,79]

In this case area 4 is divided into two bands, IV and V. They are dissimilar as regards both functional organization of the sensory cortex and threshold to electrical stimulation, a difference well known in the macaque (Hines). It is interesting in this connection to note that for cytoarchitectonic reasons von Economo[40,41] divides Brodmann's[5] area 4 into two bands, FA and FAγ, which may correspond to our Bands IV and V in the chimpanzee.

The electrical excitability of the immediate postcentral band, VI, is

entirely in harmony with the findings reported in the literature concerning monkeys,[27,43,72] orang,[4] and man[44–46,59] and enough has been said of the discrepant findings in the chimpanzee. Its posterior margin conforms curiously closely to that found in man. That margin is bounded by the suppressor region, Band VII, which has its functional homologue in the brain of the macaque (area 2s). While no previous investigators have reported responses to stimulation posterior thereto the conditions have never been the same.

That responses could be elicited by electrical stimulation of the posterior parietal sensory cortex, Bands VIII and IX, was, in view of statements to the contrary in the literature, most surprising. The only definite indication that movements could be elicited from this region in the monkey is given in the drawing of C. and O. Vogt[78] (see p. 438) and from this region in man cited by Foerster.[44–46]

The failure to obtain ocular movements from this same region seems, in view of the findings in the macaque, to indicate that the stimulus here employed did act upon the cells of this region rather than upon some subjacent fiber system and, therefore, tends to strengthen rather than to weaken the importance of our findings of motor response from this region.

The posterior margin of this "excitable" or "motor" posterior parietal region may well be defined by the suppressor band, XI, which lies just outside the sensory cortex.

This suppressor band, XI, which has its counterpart in the macaque[30] lies in a region whose position and shape suggests area 19 of Brodmann. With respect to results of electrical stimulation it resembles the three suppressor bands previously described.

The major conclusion of these investigations on the chimpanzee, covering both the functional organization of the sensory cortex and the "motor" response to electrical stimulation of the same hemispheres, is that "motor" responses can be elicited by appropriate electrical stimulation from practically all parts of the cerebral cortex which, by the criteria used, can be considered "sensory," except those bands, III and VII, where stimulation produced muscular relaxation instead of contraction.

Three considerations militate against the simple statement that this "sensory cortex" is also "the motor cortex." First, there is the factual difficulty with respect to the suppressor bands, III and VII, for while a relaxation is as truly a response as is a contraction, inclusion of bands giving relaxation would bring under this caption Bands I and XI, neither of which is "sensory" and one of which at least includes points motor for the eyes. Second, responses to electrical stimulation of the parietal regions depend, as shown in the experiments here cited, upon two adjuvant factors, namely, great tension in the muscles to be moved and facilitation of the secondary type beginning with the electrical stimulation of a point giving a primary response. The third consideration is a matter of terminology. For historical reasons the term "motor" cortex now means primarily "area 4," whereas the term "sensory" cortex has come to mean that region strychninization of which produces, in

the non-narcotized state, symptoms of somatic sensory excitation.[24,25] Had the term "sensory" cortex been coined to designate that part of the cortex in which most discrete somatotopic localization is found,[3,63] it would have had a much more restricted meaning, implying the postcentral region, par excellence. For all these reasons it seems best to state the conclusions as follows.

Conclusions

Appropriate electrical stimulation of the cerebral cortex of the chimpanzee under moderate Dial narcosis exhibits facilitation and extincton of motor response. Although their relation within a single response is more complex than in the macaque their dependence upon the parameters of stimulation is the same.

Stimulation with parameters selected to avoid facilitation and extinction, or to obtain facilitation when necessary to elicit responses from regions otherwise unresponsive, reveal the following:

With the exception of two narrow bands practically all parts of the cortex which, by the criterion here used, can be considered sensory yield contraction of skeletal muscles.

These two bands, III and VII, within (and two others, I and XI, just without) the sensory cortex yield (i) suppression of motor response to cortical stimulation, (ii) suppression of motor after-discharge following cortical stimulation and (iii) relaxation of skeletal muscles.

With the exception of the parietal area, which requires facilitation and existing muscular tension to exhibit motor response to cortical stimulation, the bands of the cortex revealed by electrical stimulation and motor response are identical with the bands revealed by strychninization and recording of electrical activity.

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FUNCTIONAL INTERDEPENDENCE OF SENSORY CORTEX AND THALAMUS*

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IN A RECENT paper on "Physiological delimitation of neurones in the central nervous system"[9] the following statement was made: "This cortex showed, naturally, very little spontaneous activity due to the extirpation of its thalamus, by which procedure the cortico-thalamic-cortical circuits, necessary for the maintenance of the normal electrocorticogram, were destroyed." Substantiation would have been out of place there; we, therefore, present it here.

Monakow[12] was one of the first to recognize the existence of numerous corticothalamic neurones besides the well-known, sensory thalamocortical ones. In 1895[13] he gave the first diagram showing the anatomical interrelation of the cerebral cortex and the thalamus. Subsequent experimental and clinical neuroanatomy have substantiated this finding (Kölliker, Déjérine, Flechsig, Bechterew, Minkowski, Poljak, Walker, Levin and others).

Head and Holmes[11] were the first to assign a function to the corticothalamic neurones, namely inhibition of the "essential centre" of the thalamus, *i.e.*, of its medial nuclei which they presumed subserve the affectional side of sensation.

In previous papers the present authors[3,4,8] have given the first experimental evidence of the functional interrelation of sensory cortex and thalamus, namely, that local strychninization of each of several of the constituent areas of the sensory cortex results in activation, "firing," of the sensory thalamic nuclei (as evidenced by the appearance of strychnine spikes in the electrothalamogram) and local strychninization of a sensory thalamic nucleus "fires" the corresponding subdivision of the sensory cortex.

Other experiments gave evidence that excitation of the sensory cortex excites the sensory thalamic nuclei[1,2,6] and excitation of these nuclei excites the sensory cortex. In subsequent experiments it was established that local strychninization, or mechanical or electrical stimulation of a certain area of the sensorimotor cortex, area 4-s, results in a typical temporary suppression of electrical activity of other portions of the sensory cortex (area 4) and that this suppression is brought about via the caudate nucleus and the thalamus.[8] Thus the operation of cortico-subcortico-cortical circuits was established. In this paper evidence will be presented that the "normal" electrical activities of the cortex and of the thalamus, even in the narcotized animal and without any especial excitation depend upon the intactness of their mutual connections, the cortico-thalamo-cortical circuits.

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METHODS

All experiments were performed on monkeys (*Macaca mulatta*) anaesthetized with Dial.* The electrical activity of the cerebral cortex was taken directly from its surface, *i.e.*, the electrocorticogram (ECG) recorded, with bipolar electrodes feeding into a suitable amplifier and recorded with cathode ray oscillograph, Westinghouse oscillograph or inkwriter oscillograph (Grass). For simultaneous recording of the electrical activity of subcortical structures concentric needle-electrodes were used and their position checked at necropsy.

RESULTS

The problem is: What structures are necessary for the maintenance of the "spontaneous" electrical activity of the cerebral cortex, *i.e.*, for the normal ECG, in the narcotized monkey?

It should be noted first that so long as the blood pressure or at least the blood supply of the brain can be maintained in the "decapitate" preparation, the electrical activity of the cortex appears unaltered. The term "decapitate" as here used means that the cord has been severed at the level of the foramen magnum. The same is found in the "decerebrate" preparation even when the level of section of the brain stem is above the superior colliculus. Figure 1 exemplifies these results.

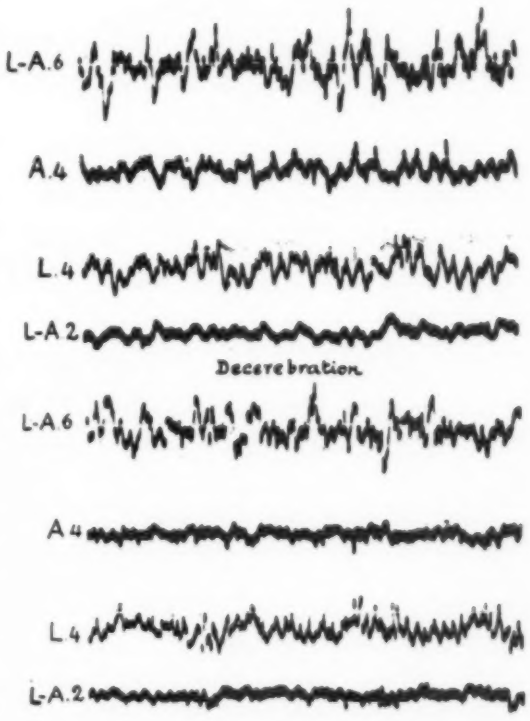

FIG. 1. April 11, 1938. *Macaca mulatta*. Dial. Curare. Artificial respiration. ECGs taken before and 1 min. after decerebration.

Similarly, division of the corpus callosum can be accomplished without significant alteration of the spontaneous electrical activity of the cortex. The only difficulties are technical. In our most successful attempts, the calvarium was removed bilaterally and section was made by retracting the hemisphere opposite to the one recorded so that the slight trauma produced by retraction would not cause a diminution of activity of the area to be examined.

Even in such a preparation the electrical activity remains if a small area from which to record is left intact and the rest of the cortex of the same hemisphere is removed subpially, *i.e.*, by suction, leaving the blood supply to the remaining structures. Figure 2 shows the electrical activity recorded from the arm area 4 before and after subpial resection of various other

* The Dial was kindly put at our disposal by the Ciba Co.

cortical areas one after another until virtually all that remained on the convexity was the area recorded. This finding is confirmed by sectioning the cortico-cortical connections. Although the incisions pass all the way to the nucleus caudatus medially and the putamen laterally the activity is not significantly altered. Finally, gross lesions of the basal ganglia do not appreciably diminish the activity of the cortex. In fact, as has been stated elsewhere, even small lesions of the nucleus caudatus are followed by augmentation of activity for a matter of hours. Here it should be stated that in no one animal have all of these lesions been made. However, so many combinations have been made that it is difficult to imagine any way in which this could lead to a false conclusion.

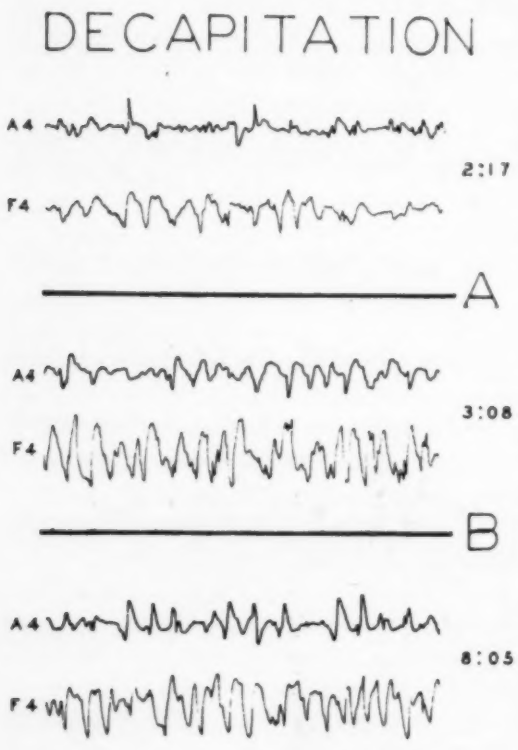

FIG. 2. Nov. 17, 1939. *Macaca mulatta*. Dial. Decapitation at 11:22.
15 mm. per sec. 6.5 mm. = 20 μV.
A = Removal of postcentral sensory arm cortex by suction at 2:23.
B = Stepwise removal of all sensorimotor cortex except A4 and F4 (3:15 to 7:45). (Reduced to ⅔ size)

The results can, therefore, be summarized briefly. Essentially normal electrocorticograms can be obtained subsequent to any lesion or combination of lesions that does not compromise the cortical area whose activity is recorded, the thalamus or their interconnections.

When one asks not what may be destroyed and leave normal electrical activity in a cortical area, but what lesions prevent it, the following results are significant.

Obviously the entire cortex cannot be destroyed and an ECG obtained. In previous publications from this laboratory[5,7,14] it was shown that laminar thermocoagulation of all layers external to that of the large and giant pyramidal cells while temporarily obliterating almost all activity did not produce any permanent deficit or alteration, whereas thermocoagulations which included the layer of large and giant pyramidal cells and left only the polymorphic layer produced a permanent and profound reduction of electrical activity.

If one makes a lesion in the ventro-lateral nuclei of the thalamus which have been shown to be sensory[10] and which interrelate with the sensory cortex as mentioned above, then the corresponding subdivision of the sensory cortex shows a diminution of its electrical activity. Here it is important to

note the lack of change in the ECG of other areas. Figure 3 exemplifies both findings.

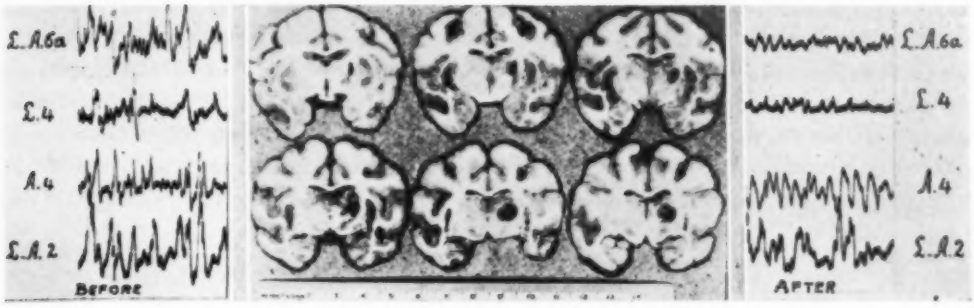

FIG. 3. March 18, 1938. *Macaca mulatta*. Dial. ECGs taken of right hemisphere before and 1 hour and 40 min. after lesion of leg-nuclei of right thalamus.

Next, if one severs the connections of the cortex and thalamus, by deep undercutting, the cortical activity immediately disappears. This is seen in Fig. 4.

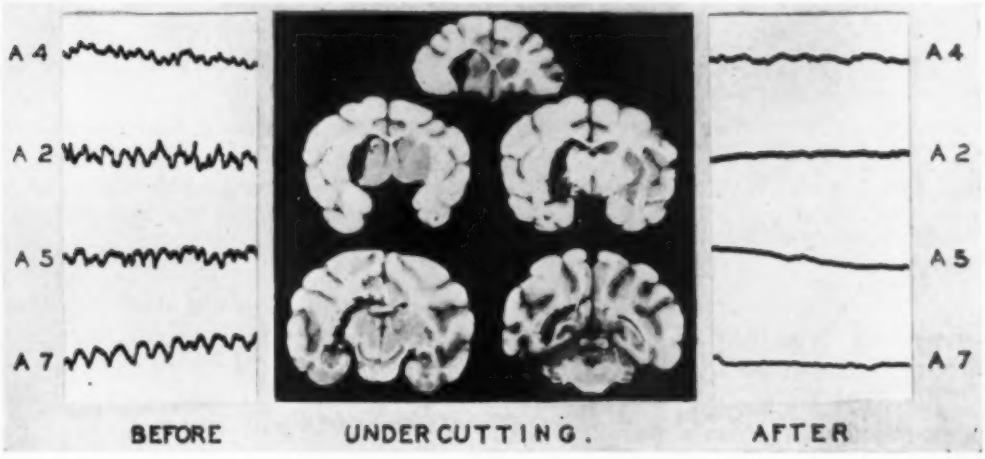

FIG. 4. Feb. 1, 1937. *Macaca mulatta*. Dial. ECGs taken before and after deep undercutting of right sensori-motor cortex.

It should be added that such deep undercutting leaves the blood supply of the cortex so good that its threshold to electrical stimulation, judged by the appearance of electrical after-discharge, is unaltered. One other result of such a lesion is of interest here, namely, that the electrothalamogram (ETG), in spite of the intact afferent tracts from lower levels, shows an almost equal reduction.

No animal with such an extensive undercutting has been kept for a

sufficient length of time to determine how much activity might return. With a small lesion cortico-cortical connections might well be expected to play an important role in restoration of activity. Even in the acute experiment this supposition finds support, for the ECG of an area locally undercut is never completely inactive.

Nevertheless it was considered worthwhile to know what permanent alterations would follow from a relatively small undercutting of the sensory cortex. A lesion was therefore made under the medial half of arm area 4 and extending partly under leg area 4. This animal was kept for over $2\frac{1}{2}$ years to allow complete retrograde degeneration to obliterate all cells whose axones

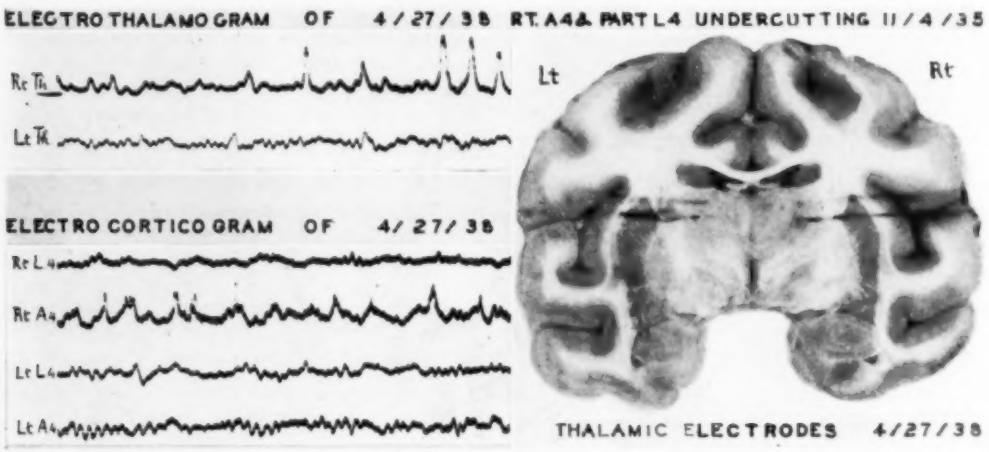

FIG. 5. April 27, 1938. *Macaca mulatta*. Dial. Last ECGs taken 2.5 years after undercutting of A4 and part of L4 of right hemisphere. Animal at rest with its eyes closed showing alpha frequency circa 10 per sec. in normal structures.

had been severed. When the skull was opened the dura was not adherent over the areas to be studied. In order to be certain that the findings were not unduly influenced by anaesthesia the cortical records were first obtained under light anaesthesia with the animal's head securely clamped. Thalamic electrodes, with shields grounded, were then inserted and records were made from them. The monkey was allowed to come out from under anaesthesia gradually and continued to lie quietly on the table. The records then obtained show an alpha rhythm from normal cortex and thalamus but abnormal activity from the cortex and thalamus which had been separated by undercutting $2\frac{1}{2}$ years before. Figure 5 shows the location of the thalamic electrodes, and the last records obtained from this monkey resting undisturbed with his eyes closed.

DISCUSSION

Inasmuch as small lesions involving the deeper layers of the sensory cortex or the sensory nuclei of the thalamus or the tract through which these

are mutually related cause permanent local abnormality and loss in spontaneous electrical activity of the corresponding parts of both cortex and thalamus, whereas no lesion or combination of lesions elsewhere does so it seems appropriate to regard these, and only these, structures as essential to that activity. Since excitation of either of these gray masses has already been shown to excite the other and neither can support "spontaneous" activity in isolation from the other, it is clear that the activity of either depends upon activity reaching it from the other. That is, for spontaneous activity, the sensory cortex and the optic thalamus are functionally interdependent.

Summary

When fall of blood pressure or other impairment of circulation is avoided, decapitation, decerebration, section of the corpuscallosum, and subpial resection of the entire cortex of the convexity of the hemisphere, save that area to which electrodes are applied, one and all leave the electrical activity unaltered. So also does destruction of sensory thalamic nuclei other than that corresponding to the subdivision of the sensory cortex whose activity is being recorded. Lesions of the basal ganglia have little or no effect on the activity of the sensory cortex or of the corresponding thalamic nuclei, except for a transient increase following injury to the nucleus caudatus.

On the other hand a lesion of the sensory thalamic nucleus corresponding to a cortical area under investigation and a lesion of the cortical area corresponding to a sensory thalamic nucleus under investigation, each results in a permanent abnormality and diminution of spontaneous electrical activity on the other. The same obtains when their mutual connections are interrupted.

Laminar thermocoagulation of the superficial layers has previously shown that the layers of the cortex external to that of the large and giant pyramidal cells are not necessary to the maintenance of the normal ECG.

It is therefore concluded that, for the maintenance of the normal spontaneous electrical activity of the sensory cortex and of the corresponding sensory thalamic nuclei, the essential structures are the deeper layers of the cortex, the corresponding thalamic nuclei and their mutual connections, and, hence, that for this activity the sensory cortex and the ventro-lateral thalamic nuclei are functionally interdependent.

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SUPPRESSION OF MOTOR RESPONSE OBTAINED FROM AREA 4 BY STIMULATION OF AREA 4s*

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IN 1934 during the investigation of facilitation and extinction of motor response to cortical stimulation[1,2,3,6,7,8,9,12,17,18,19] due to antecedent stimulation of neighboring foci a new phenomenon appeared when the antecedent stimulation was in either the frontal part of area 4 or in the posterior part of area 6. When stimulation of this region preceded by several minutes the testing stimulation of area 4 no response to that stimulation of area 4 was obtained. Careful examination of the records showed frequently a slight lowering of the base line, suggesting a relaxation of the extremity. During mapping of the functional organization of the sensory cortex,[10] the region from which the new phenomenon could be elicited was discovered to give, upon local strychninization within it, a diminution of the electrical activity of area 4. The area yielding these phenomena, "suppression of motor response" and "suppression of electrical activity," was designated area 4s and was found to coincide with the strip of cortex from which Dr. Marion Hines obtained, by electrical stimulation, a cessation of movements and a relaxation of contracted muscles[14] and ablation of which by the same author produced spasticity.[13]

In a previous publication on the functional organization of the sensory cortex[10] it was shown that the "firing" of one area by another was dependent upon cortico-cortical connections which remained intact when the cortex was severed from deeper structures, by deep undercutting, whereas, in a subsequent publication,[11] it was demonstrated that the suppression of electrical activity of area 4 by strychninization of area 4s was dependent upon a one-way circuit from area 4s to the nucleus caudatus, thence to the thalamus and so to the cortex. As it was hoped, at that time, that the analogous phenomenon, the suppression of motor response, might readily be traced either in the cortico-cortical system or, more probably, through the corresponding deeper structures the publication of a full description of the phenomenon was delayed until the experiments could be performed to demonstrate its circuit. This proved more difficult than was expected, wherefore it was deemed wisest to write the present article in which the phenomenon is fully described, the distinction between it and the extinction of motor response made clear and, finally, the attempts to trace its path through the central nervous system are recorded together with the reason for each experiment.

* Aided by a grant from the Fluid Research Funds of the Yale School of Medicine.
† Deceased June 9, 1940.

METHODS

All experiments were performed upon monkeys (*Macaca mulatta*) anaesthetized with Dial* (0.45 cc. per kg. body weight, ½ the dose intraperitoneal, ½ intramuscular). The cortex was exposed and stimulated with thyratron impulses delivered through Ag-AgCl electrodes. Responses were recorded isotonically on a smoked paper kymograph. When necessary these procedures were supplemented by recording the electrical activity of various structures of the central nervous system. This was done with moving paper camera and 4-channel Westinghouse oscillograph with appropriate amplifiers.

Lesions were made chiefly by a small blade mounted on a mechanically controlled needle which could be thrust into the brain at any required angle to any required depth, there rotated and then returned to the original position before retraction, thus minimizing the lesion of approach.

Brains with lesions were taken up into a sarcophagus with agar (of approximately the same consistency as the brain), extruded against an angular mount, sectioned with a spatula into slices either 1 or 2 mm.,[4] and photographed.

RESULTS

Stimulation of the cerebral cortex with a manually applied electrode by well controlled thyratron impulses discloses that the threshold rises fairly

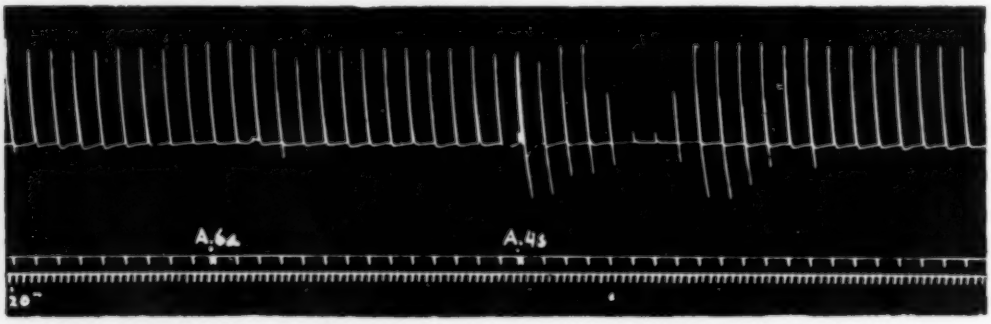

FIG. 1. Sept. 27, 1938. *Macaca mulatta*. Dial. Electrical monopolar stimulation (Thyratron) of A4 focus once every minute (5 sec.-0.6 μF.-40 per sec.-V.D. 1200). Extension of wrist. Electrical stimulation of A6a focus gives no suppression. Electrical stimulation of A4s focus (6 sec.-1 μF.-40 per sec.-V.D. 7000) gives suppression. Time line = 20 sec.

abruptly as one passes from the posterior to the more anterior part of area 4. This change is encountered in about the middle of area 4 and is of the order of a 100 per cent rise in threshold. If the impulses are relatively long this threshold is not very different from that of area 6, but between area 4 and area 6 lies a narrow band of cortex (2 or 3 mm. wide and running dorsoventrally through the anterior part of the superior precentral sulcus) where stimulation fails to elicit any contractions of muscles until such voltages are reached that spread of current must have involved the adjacent areas. This band of cortex from which no contraction of muscles is elicited is the strip of Marion Hines,[13,14] area 4s, *i.e.*, the strip from which one obtains a suppression of electrical activity of area 4. But the stimulation of area 4s, although it fails to elicit contraction, has (as Marion Hines has shown) definite effects which indicate that it has excited the cortex.

* We wish to thank the Ciba Co. for kindly putting the Dial at our disposal.

Figure 1 shows the effect with which this paper deals, namely, the suppression of motor response to electrical stimulation of a motor focus of area 4 by antecedent stimulation of area 4s. If one stimulates a focus of area 4 once a minute with such a stimulus that neither facilitation nor extinction disturbs the amplitude of the successive responses and then one stimulates area 4s the responses to the area 4 stimulation diminish or disappear.

Figure 2 shows that this suppression of motor response can be brought about by chemical or mechanical as well as by electrical stimulation of area 4s, and therefore cannot be due to inadvertent stimulation by spread of current to area 4 or to subjacent fibre tracts—a conclusion confirmed by thermocoagulation of the entire thickness of the cortex which prevents suppression.

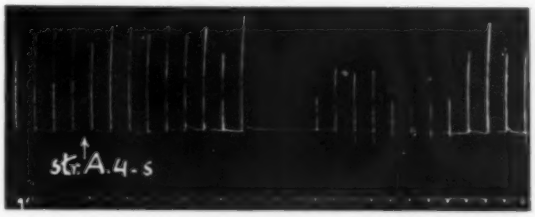

FIG. 2. April 13, 1939. *Macaca mulatta*. Dial. Electrical stimulation once every minute of focus of A4 for extension of fingers (5 sec.-0.5 μF.-40 per sec.-V.D. 3600). Strychninization of A4s. Note 9 min. latency, and bimodal suppression.

Several characteristics of this suppression of motor response are noteworthy: (i) The suppression has a remarkable latency after electrical stimulation. The duration of latency most frequently encountered is 4 min., but latencies as short as 2 min. or as long as 12 min. have been obtainable by use of light anesthesia and strong stimulation of 4s for the shorter, and deep anesthesia and weak stimulation of 4s for the longer latencies. After mechanical stimulation the latency is usually short, whereas after strychninization, measured from the moment of application, the latencies are usually from 12 to 20 min. (ii) The suppression lasts several minutes. Suppression of motor response to 6 or 7 stimulations at one-minute intervals is ordinarily encountered. With weak stimulation of area 4s the duration is usually shorter. Suppressions lasting 20 to 30 min. are not uncommon with strong stimulation of area 4s in deeply narcotized animals. (iii) The suppression, if long, is frequently bimodal. Longer suppressions are occasionally trimodal. (Cf. Fig. 2, 5 and 7.) (iv) The suppression is accompanied by a change in muscle tension. During the suppression, sometimes starting before noticeable change in amplitude and sometimes lasting after amplitude has returned to its original level, there is a slight fall in the base line provided there is tension in the direction of gravity in the thread to the tambour in the resting position, thus indicating a loss of tension of the muscles supporting the extremity. Moreover, the last responses before, and several after the diminution (or absence) of amplitude look atonic and frequently graph the relaxed condition of the muscles by falling past the resting position. (Cf. Fig. 1 and 2.) (v) The suppression cannot be reinduced immediately. As these experiments were tedious attempts were made to find out how soon it was advisable to attempt to repeat the experiment. After a prompt, short suppression induced by me-

chanical stimulation of 4s, 15 to 20 min. suffices but with a typical 4-minute latency, 6-minute suppression following electrical stimulation of 4s, the experiment usually fails if attempted within 35 min. and succeeds at the end of 45 min.

The second group of findings are those that separate the suppression of motor response from the extinction of motor response to which it bears only a superficial resemblance.

The first, and obvious, difference is that the only part of the so-called motor cortex from which the suppression of motor response to cortical stimulation of any motor focus of area 4 can be obtained is area 4s; whereas extinction is obtained maximally by antecedent stimulation of one and the same focus and can be obtained by antecedent stimulation elsewhere only if the disturbance (notably, in the form of an after-discharge) actually involves the structures excited by the test stimulation. Whether such is the case when 4s stimulation produces unresponsiveness to stimulation of area 4 can be examined by electrical recording of the activity of the focus of area 4 to which the test stimulus is to be applied. The experiment proves that suppression of motor response can be elicited with no trace of after-discharge in any part of the cortex.

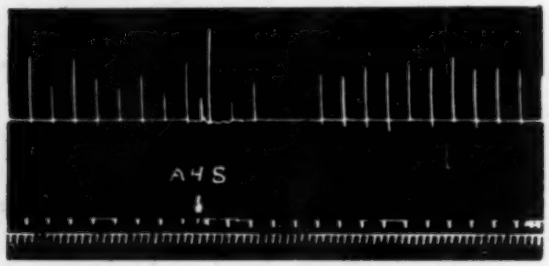

FIG. 3. Oct. 4, 1938. *Macaca mulatta*. Dial. Flexion of fingers. Stimulation of A4s (5 sec.-1 μF.-50 per sec.-V.D. 6600) at posterior margin of A4s. Note small primary response. The responses to the following A4 stimulation (5 sec.-0.5 μF.-50 per sec.-V.D. 3600) show (i), facilitation, (ii), extinction, (iii), return, then suppression.

The second difference between these phenomena is also essentially spatial. Except where antecedent stimulation has been so extreme as to initiate an after-discharge so violent as to spread across the functional boundaries between the face-, arm- and leg-subdivisions, extinction has always remained localized to the subdivision stimulated; whereas the suppression of motor response to stimulation of a motor focus of, let us say, A4 can be elicited by antecedent stimulation of L4s, A4s, F4s on the same hemisphere and even, although less markedly, by stimulation of 4s on the opposite hemisphere.

The third difference is temporal. Except for the belated components of extinction due to the slow progress of a persistent after-discharge, which have been carefully excluded, extinction lasts a matter of one or two minutes at the most, whereas suppression has a latency of some four minutes. To make perfectly certain of the temporal separation in a single suppression one has only to place the 4s electrodes on the posterior margin of area 4s of the same subdivision as that of the area 4 focus used in testing and to increase the voltage of the area 4s stimulus sufficiently so that spread of current stimulates the more anterior part of area 4. One obtains then, by proper

timing, facilitation to the first test stimulus, extinction to the second, no change to the third and suppression to the subsequent stimulations for the next several minutes. Figure 3 demonstrates this finding.

It is appropriate here to record another finding. At the suggestion of John Hamilton of this laboratory that the suppression of motor response from what was intended for stimulation of area 4s might be merely the effect of simultaneous stimulation of area 4 and area 6, two monopolar electrodes were placed one in area 6, immediately anterior to 4s, and the other in area 4, immediately posterior to area 4s and these were connected to the thyratron used for 4s stimulation. These monopolar electrodes were scarcely 3.5 mm. apart. Yet, even with slightly higher values of stimulation than sufficed in area 4s no suppression of motor response occurred. Instead, there was a primary response to the conjoined stimulation, followed by the facilitation and subsequent extinction of response to area 4 stimulation.

Inasmuch as the suppression of motor response was not restricted either to responses elicited from one part of the cortex, or to responses of a single

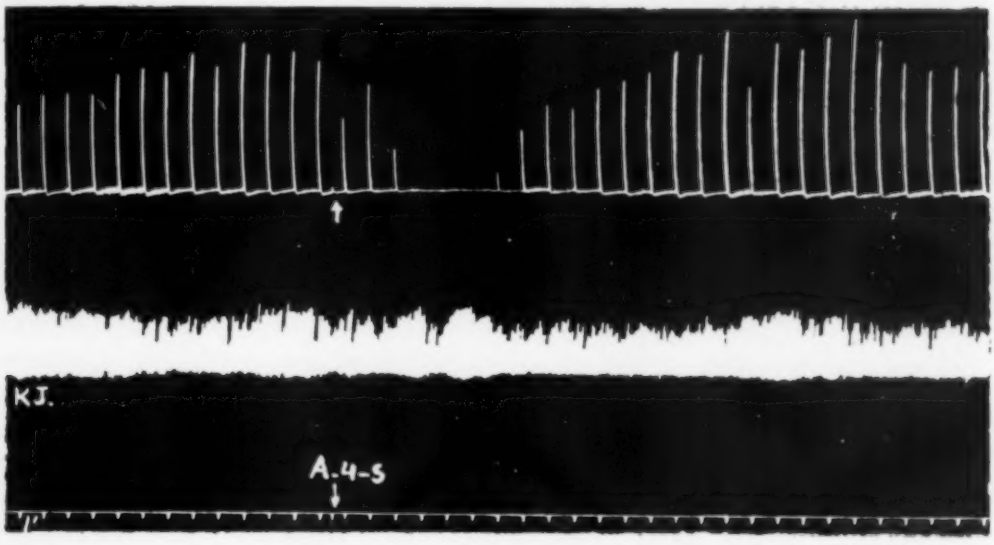

FIG. 4. March 7, 1939. *Macaca mulatta*. Dial. A4 = 5 sec.-0.5 μF.-50 per sec.-V.D. 800. A4s = 5 sec.-1 μF.-50 per sec. V.D.1600. Time line = 1 min. Upper record, extension of wrist in response to stimulation of A4 focus. Lower record, K.J. unaffected during suppression of motor response to cortical stimulation.

part of the body, the question at once arose as to whether it might not be due to an induced systemic alteration of circulation, respiration or metabolism which, in turn, produced a completely generalized rise in threshold either throughout the central nervous system or throughout the musculature. To settle this question it seemed simplest to examine a response which was initiated by other than cortical stimulation. The knee jerk in response to weak mechanical stimulation of the patella tendon every 4 sec. was, therefore, recorded before, during and after a suppression of motor response. Figure 4 shows the result of this experiment. The knee jerk is not altered.

Thus one has to look for a disturbance in restricted parts of the central

nervous system and to attempt to trace its course from the area stimulated —area 4s—to the structures involved in the response to the testing stimulation of area 4. The simplest possible path which might be involved is a cortico-cortical connection from area 4s to area 4. However, section by a deep incision down to the nucleus caudatus does not prevent suppression. Figure 5 shows the suppression elicited after such a section.

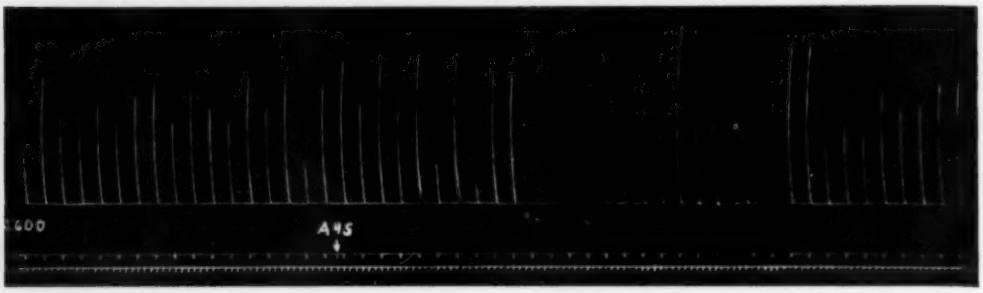

FIG. 5. Oct. 10, 1938. *Macaca mulatta*. Dial. Suppression of motor response at 9:05 P.M. (A4s = 5 sec.-1 μF.-40 per sec.-V.D. 5600. A4 = 5 sec.-0.5 μF.-40 per sec.-V.D. 2600) subsequent to large lesion of nucleus caudatus at 5:00 P.M. and incision 13 mm. deep between A4s and A4 at 7:40 P.M.

Since one had to look, therefore, into cortico-subcortical connections, and since among these the cortico-caudate projection was known to be necessary for the suppression of electrical activity of area 4 upon strychninization of area 4s, an attempt was made to pith the nucleus caudatus by a frontal approach. This lesion prevented the suppression, but at autopsy, although the nucleus caudatus was extensively injured, the blade had been at such an angle as to sever the descending fibres from area 4s, and no conclusion could be drawn as to which part of the lesion was significant in the prevention of suppression.

Therefore, a small undercutting of 4s was made. This simple lesion prevented suppression and demonstrated that the disturbance necessary for suppression required a descending track. This is the direct proof of what one could never legitimately infer by exclusion from the results of severance of cortico-cortical connections.

Next a lesion was made in the nucleus caudatus without injury to the cortico-caudate fibres external to the caudate, by approaching it at another angle. The suppression of motor response was obtained again. Figure 6 shows the lesion and the suppression. This lesion is rather small but was sufficient to prevent any suppression of electrical activity of area 4 upon strychninization of area 4s even after the suppression of motor response had again been elicited after the lesion. Larger lesions of the nucleus caudatus also failed to prevent the suppression of motor response. One is therefore forced to conclude that the nucleus caudatus which is necessary for the suppression of electrical activity is either not necessary for the analogous suppression of motor response or that remaining parts of the nucleus caudatus, although

insufficient for suppression of electrical activity of area 4, are still sufficient for suppression of motor response.

It seemed advisable in any case to exclude other parts of the same system,

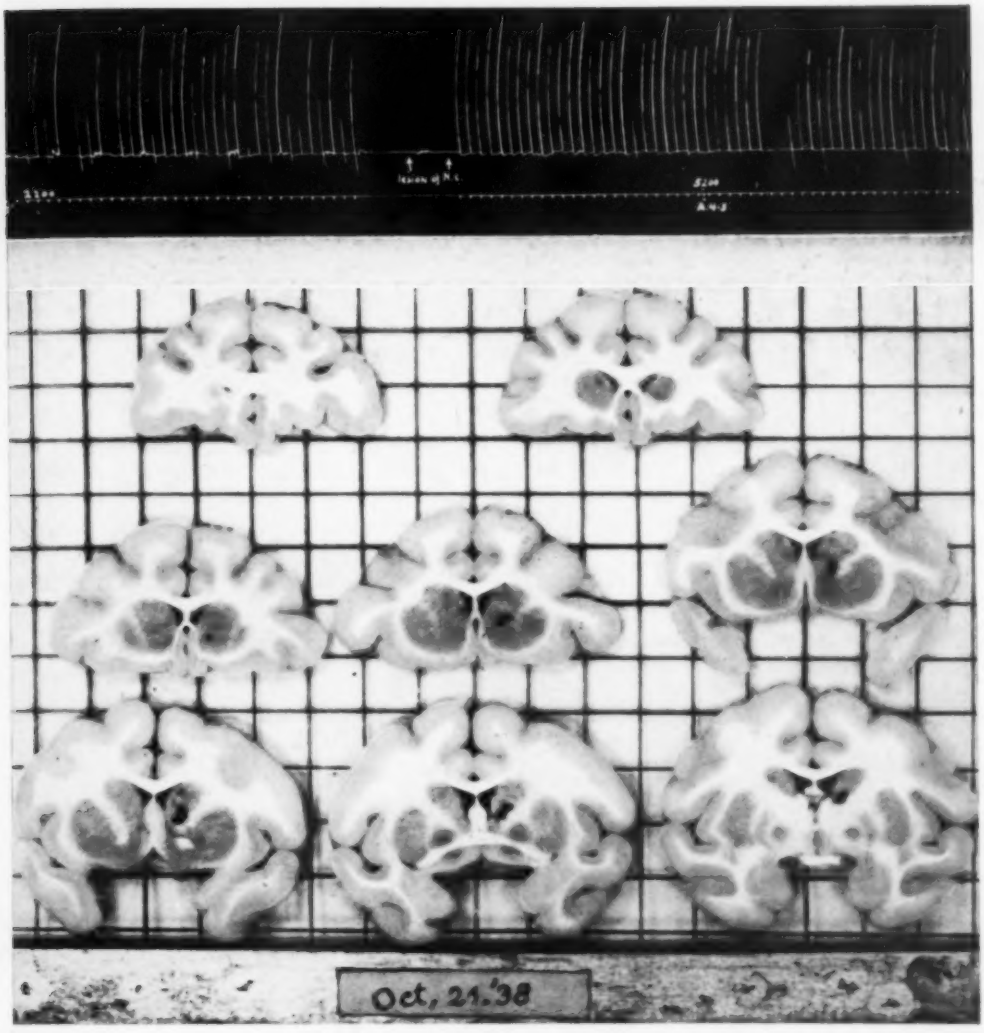

FIG. 6. Oct. 21, 1938. *Macaca mulatta*. Dial. Extension of fingers. Continuous record. "Release" of motor response and suppression of motor response after lesion of nucleus caudatus which prevented suppression of ECG of A4. A4 = 5 sec.-0.6 μF.-40 per sec.-V.D. 2200. A4s = 6 sec.-1 μF.-40 per sec.-V.D. 5200.

i.e., basal ganglia, by a similar procedure. Therefore a massive lesion was made in the putamen and the suppression was again elicited. The same obtains with respect to the globus pallidus and the thalamic nuclei (see Fig. 7).

As the descending system from the cortex anterior to area 4 is said to be traceable by myelin degeneration as far as the pons varolii but not below it

and as it is said to be connected there with the substantia nigra and the pontine nuclei,[16,20] these possible paths were investigated.

First, to exclude any influence of the opposing half of the brain, the corpus callosum, anterior commissure and commissura mollis were divided. Suppression remained. Second, the substantia nigra was pithed bilaterally.

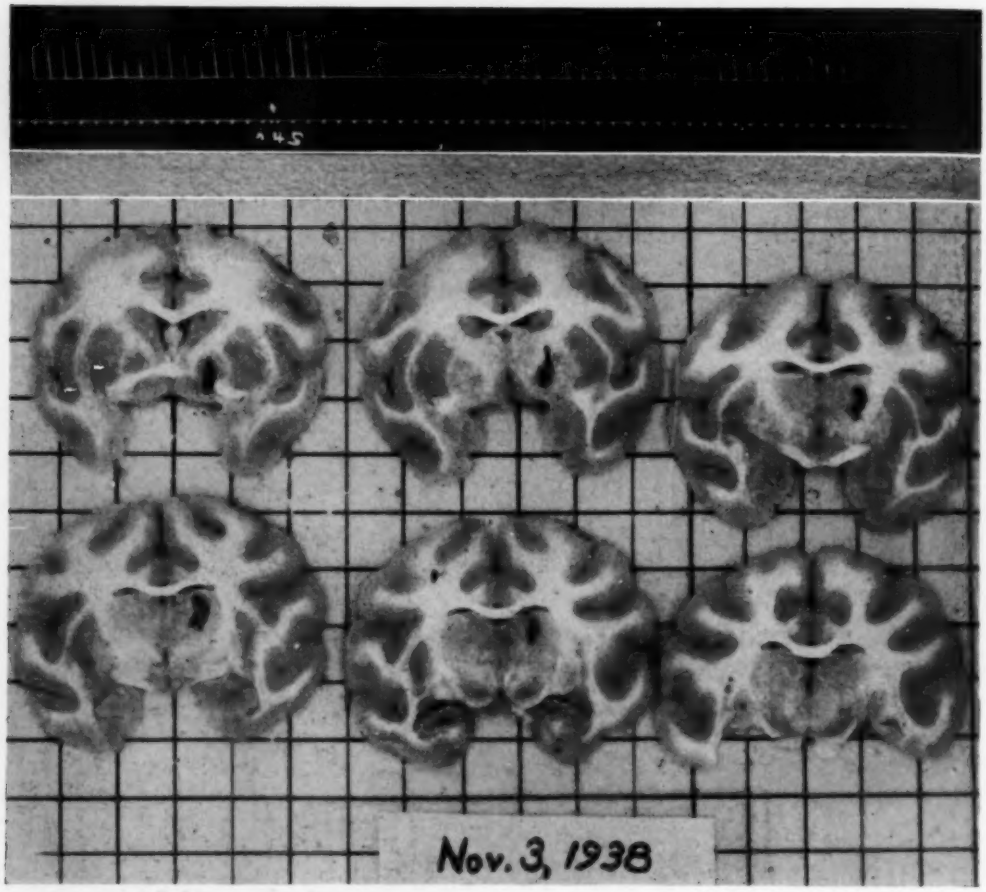

FIG. 7. Nov. 3, 1938. *Macaca mulatta*. Dial. Flexion of left wrist. Suppression of motor response present after lesion (at 8:40 P.M.) of palladum and thalamic nuclei (arm and leg) A4 = 5 sec.-0.5 μF.-40 per sec.-V.D. 2500. A4s = 5 sec.-1 μF.-40 per sec.-V.D. 7500. (10:59 P.M.). Time line = 1 min.

Figure 8 shows the results of the operation. Suppression of motor response could still be elicited from area 4s. Finally, the cerebellum was removed. Figure 9 shows the subsequent suppression of motor response.

These statements must not be taken to mean that suppression could always be obtained in each type of experiment. In fact, an experiment sometimes had to be performed on three or even four animals before it could be established that suppression could still be elicited after a lesion of the type

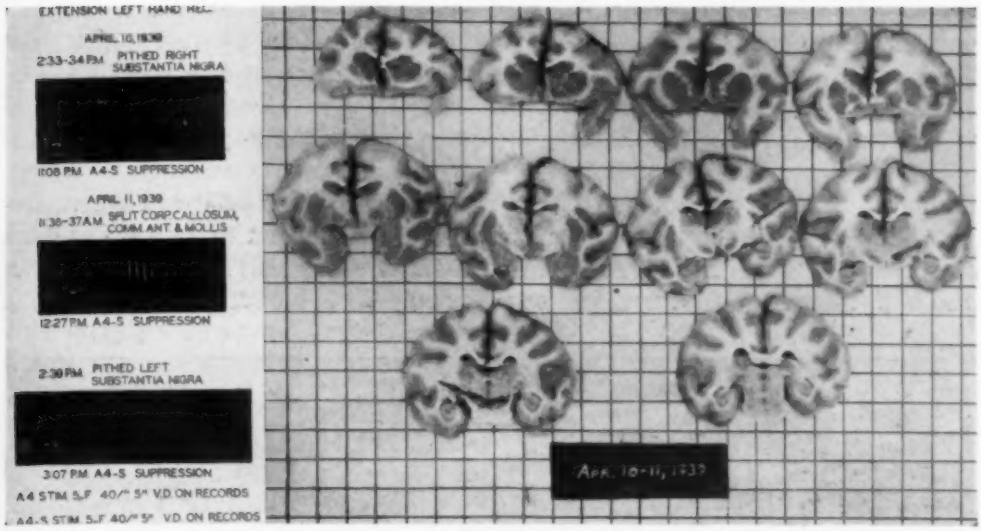
EXTENSION LEFT HAND
APRIL 10, 1939
2:33-34 P.M. PITHED RIGHT SUBSTANTIA NIGRA
11:08 P.M. A4-S SUPPRESSION
APRIL 11, 1939
11:36-37 A.M. SPLIT CORP CALLOSUM, COMM ANT & MOLLIS
12:27 P.M. A4-S SUPPRESSION
2:38 P.M. PITHED LEFT SUBSTANTIA NIGRA
3:07 P.M. A4-S SUPPRESSION
A4 STIM S-F 40/" 5" V.D. ON RECORDS
A4-S STIM S-F 40/" 5" V.D. ON RECORDS
APR. 10-11, 1939

FIG. 8. *Macaca mulatta*. Dial. Suppression of motor response after bilateral lesions of substantia nigra and splitting of brain. For parameters of stimulation see records.

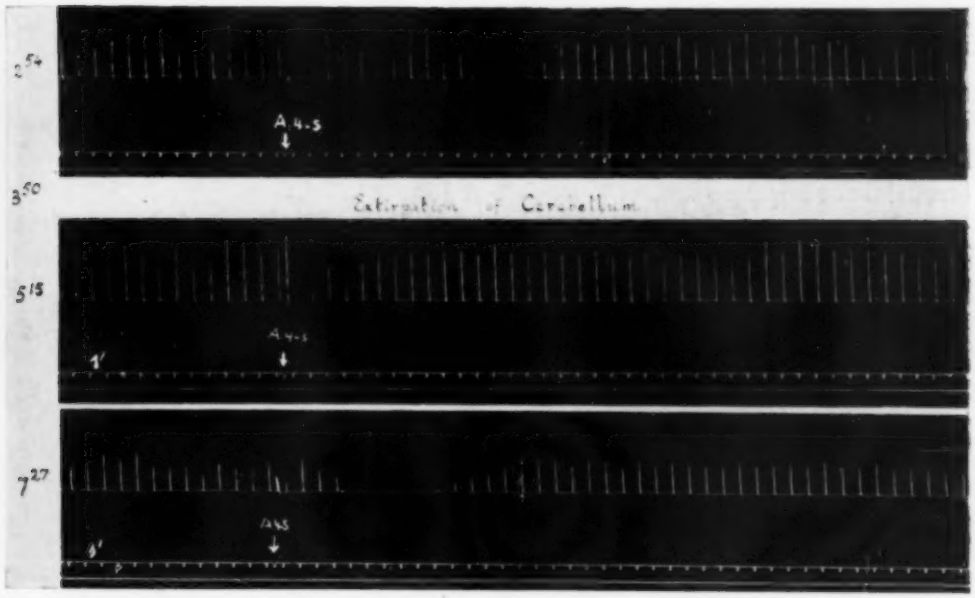

FIG. 9. Feb. 22, 1939. *Macaca mulatta*. Dial. Record 1. Suppression of motor response elicited at 3:50 (A4 = 5 sec.-0.5 μF.-50 per sec.-V.D. 2700. A4s = 7.5 sec.-1 μF.-50 per sec.-V.D. 5400). Record 2. Poor suppression elicited shortly after total extirpation of cerebellum at 5:15 when threshold was high (A4 = 5 sec.-0.5 μF.-50 per sec.-V.D. 4100. A4s = 7.5 sec.-1 μF.-50 per sec.-V.D. 8200). Record 3. Good suppression at 7:27 when threshold had returned to original value (A4 = 5 sec.-0.5 μF.-50 per sec.-V.D. 2800. A4s = 7.5 sec.-1 μF.-50 per sec.-V.D. 5600).

in question. The reason for not reporting the negative cases even when they outnumbered the positive cases, is that one can deduce anything only from a positive finding. Failure to find suppression subsequent to a lesion may be due to any number of causes, but the presence of suppression after a lesion destroying some structure clearly indicates that that structure is not necessary for suppression. On looking over early records of such failures to obtain suppression it became clear that these failures usually occurred when, following the lesion, the threshold to electrical stimulation of area 4 has been raised and before it has returned to normal. Therefore, in later experiments, the suppression of motor response was sought about every hour until the

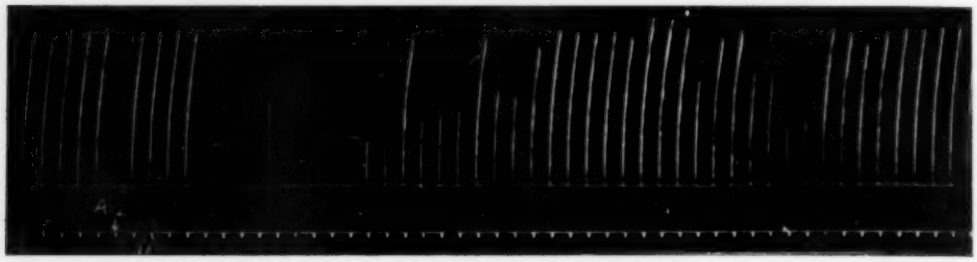

FIG. 10. April 19, 1939. *Macaca mulatta*. Dial. Suppression of motor response to electrical stimulation (0.5 μF.-50 per sec.-V.D. 1300) of A4 produced by stimulation (1 μF.-50 per sec.-V.D. 2600) of A2. Time line = 1 min.

threshold had been returned to its original value for at least one hour. This greatly reduced the number of animals that would have been required otherwise and proved that the return to approximately pre-lesion threshold was essential for suppression. (Cf. legend of Fig. 9.)

After these experiments had been completed it was discovered that stimulation in the postcentral gyrus also produced a suppression of motor response, and still more recently the same has been encountered from area 8 and from a strip of cortex lying immediately occipital to the sensory cortex. Figure 10 shows the suppression of motor response to electrical stimulation of area 4 by antecedent stimulation of the postcentral gyrus.

DISCUSSION

The suppression of motor response to stimulation of area 4 by antecedent stimulation of area 4s has obviously raised many more questions than the investigation has been able to answer. It is well, therefore, to consider first what the experimental facts prove even if these conclusions are negative. The phenomenon itself remains interesting and can always be elicited.

First, the phenomenon has been clearly demonstrated to be something other than extinction, for the two can be separated both spatially and temporally. Second, it cannot be explained as a result of the extinction of activity in area 4 due to stimulation of 4s, for suppression of motor response occurs after a lesion of the nucleus caudatus which prevents suppression of electrical

activity of area 4. Third, its long latency indicates an indirect, or complex, "delay" path. Fourth, its long duration, indicating a continuous activity of some sort (comparable to that found for after-discharge following strong cortical stimulation), together with the even longer subsequent period during which the suppression cannot be reinitiated (comparable to the duration of extinction following such an after-discharge) indicate that one is probably dealing with an activity antagonistically related to that of the motor system excited by stimulation of area 4 rather than with extinction of activity contributory to that motor system. If these suppositions be correct it follows that one is here dealing with augmented activity in a system antagonistic to the cortico-spinal system, and it becomes proper to regard suppression of motor response as an example of "inhibition," although not in the sense in which Sherrington originally used the term to designate the relaxation of the antagonist during contraction of the reciprocally innervated agonist, since, in suppression, the relaxation involves all muscles observed. The antagonism is not between agonist and antagonist muscles but between pyramidal and extrapyramidal systems within the central nervous system and there is no evidence that activity of area 4 is antagonistic to activity of area 4s. If the antagonism is not mutual there is no reason to regard the relation between the pyramidal and extrapyramidal systems as reciprocal.

One more observation tending to the same conclusion may be cited here. In the paper concerning the motor cortex of the chimpanzee[5] it is stated that the motor after-discharge (following stimulation of areas giving motor response) was held in abeyance during stimulation of suppressor bands which have been shown to correspond to 8s, 4s, 2s and 19s in the monkey. While this finding was obtained by use of more appropriate stimulation (*i.e.*, of longer pulse form) than was used in the experiment reported here and the work was on another species, it still seems significant to mention it here because it was obtained from the bands yielding suppression of motor response as here described, and suppression of electrical activity. Another previous finding, namely, that the deeply undercut cortex could support a prolonged after-discharge, indicating the essentially cortical nature of this type of after-discharge, shows that the holding in abeyance of the motor after-discharge can only be interpreted to mean that cortico-spinal impulses continued to be delivered to the cord but that its motor horn cells were rendered unresponsive by impulses traversing other descending systems. In this case there is no possibility of extinction; *per contra*, as the stimulus is there at the time and is adequate, there is every reason to expect that some system whose activity is antagonistic to cortico-spinal activity is contemporaneously active.

Thus consideration of time relations, of co-present relaxations and of holding in abeyance of after-discharge, all indicate activity in a system antagonistically related to the cortico-spinal system, *i.e.*, "inhibition" of the response to cortico-spinal activity by contemporaneous activity of some antagonistic extrapyramidal system.

The chief difficulty with this hypothesis arises from that constancy of the knee jerk in response to weak patellar stimulation which proved that the path of the "inhibiting" disturbance was within the central nervous system. It is hard to see how the motor horn cells involved could respond normally to dorsal root stimulation and not to cortico-spinal stimulation. The difficulty may be due to insufficient knowledge of the events in the spinal cord, but until these are known the notion that suppression of motor response is a differential "inhibition" of the final common path must be held reservedly. Alternative conceptions of the site of the hypothetical "inhibition" are confronted by anatomical objections.[15]

With respect to what structures of the extrapyramidal system are involved in this suppression the seemingly most "unsuccessful" experiments are most conclusive. They have shown of one structure after another that it is not necessary to this suppression, *i.e.*, that the remaining structures are sufficient. The only structures whose destruction has yielded a seeming "success" remain of questionable importance, namely, the ansa lenticularis, the fields of Forel and the red nucleus, and inasmuch as after the lesion was made the threshold of area 4 never returned to its original value these experiments are equivocal. What remains most surprising is that suppression of motor response does not depend upon an intact nucleus caudatus, for this has been shown to be the only part of the corpus striatum to which areas 8s and 4s send axones. Here the possibility remains that remaining parts of this structure sufficed for this suppression but not for that of electrical activity.

Summary

Motor response to electrical stimulation of area 4 is suppressed (typically, after a latency of several minutes for a duration of 5 or more minutes) by electrical or other stimulation of area 4s. It cannot be initiated again for many minutes.

This suppression is brought about by stimulation of cells in area 4s and does not require stimulation of underlying fibre tracts.

It has been shown to be distinct from extinction of motor response by temporal and spatial consideration.

It depends for its occurrence upon fibre tracts descending from 4s to deeper structures and not upon cortico-cortical connections.

It is, despite its great generality, restricted in its distribution within the central nervous system, as is proved by its failure to affect the knee-jerk.

The following structures have been shown to be severally unnecessary for its occurrence: nucleus caudatus, putamen, globus pallidus, thalamus, substantia nigra and cerebellum.

It is at present best conceived as brought about by increased activity in some portion or portions of the extrapyramidal system whose activity is antagonistically related to that of the cortico-spinal system.

Finally, it can be obtained from areas 8s, 4s, 2s and 19s, *i.e.*, from all of those cortical areas from which suppression of electrical activity of the cortex

can be obtained, although its occurrence is not dependent upon the suppression of that activity.

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FUNCTIONAL ORGANIZATION OF SENSORY AND ADJACENT CORTEX OF THE MONKEY*

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IN PREVIOUS papers concerning the functional organization of the sensory cortex[7,11] the boundaries of that area have been mentioned and it has been stated that strychninization immediately outside of it failed to fire any part of it. So clear was the evidence to this effect in *Macaca mulatta* that it was used as a criterion of the sensory cortex in the researches on the chimpanzee.[2,3,4,10] The present study deals with the properties and relations of these adjacent regions and with detailed observations on constituent areas of the sensory cortex which had to be precisely determined in order to be certain as to the site of electrodes and strychninizations here employed. These experiments were interspersed among studies of the same regions on the chimpanzee's brain in which it was often possible to keep strychninization confined well within a single physiologically unique band and thus to obtain a clearer conception of what to attempt in the monkey. More precise knowledge of several areas has thus been brought to light so that, aided by occasional histological control, it is now possible to improve on the previous diagram of functional organization within the sensory cortex and to be relatively certain about cytoarchitectonic correlations.

METHODS

All experiments were performed upon monkeys (*Macaca mulatta* and one mangabey) fully anaesthetized with Dial§ (0.45 cc. per kg. body weight, ½ the dose given intraperitoneally, ½ intramuscularly). The animal's head was then secured in a head holder. Strychnine was applied to the cortex by placing thereon pieces of filter paper, 1 to 4 sq. mm., moistened with 3 per cent solution of strychnine colored with toluidine blue. All records of electrical activity were made by means of six channel Grass amplifiers and ink writers. Thirty-six electrodes—each a silver wire ending in a small silver ball—were placed on the cortex. These were arranged in groups of 6 to facilitate switching and the activity of all 36 loci was recorded before and several times after each local strychninization.

The identification of the strychninized area was made by pricking or slitting it with a sharp knife at the time the strychnine paper was removed, fixing the brain in alcohol, sectioning, and staining with toluidine blue. For the certain cytoarchitectonic identification of areas 2s and 19s with Brodmann's 2 and 19 respectively we are indebted to Dr. Gerhardt von Bonin.

RESULTS

Application of strychnine to any gray mass causes large sudden voltages

* Aided by a grant from the Fluid Research Funds of the Yale School of Medicine.
† Deceased June 9, 1940.
‡ National Research Fellow, 1940–1941.
§ We wish to thank the Ciba Co. for kindly putting the Dial at our disposal.

to appear in all places where the axones, or collaterals, of cells in the region strychninized terminate. These sudden voltages—called strychnine spikes—serve, therefore, to reveal the directed functional relation of various cortical areas.[9]

Figure 1 is a new map of the sensory cortex and adjacent regions. It is made on the basis of the previous maps of the sensory cortex and has been extended in conformity with the new findings. The numbers indicate the cytoarchitectonic areas of Brodmann[1] and the Vogts[16] but are simplified in the face region where no distinction could be made on the basis of organiza-

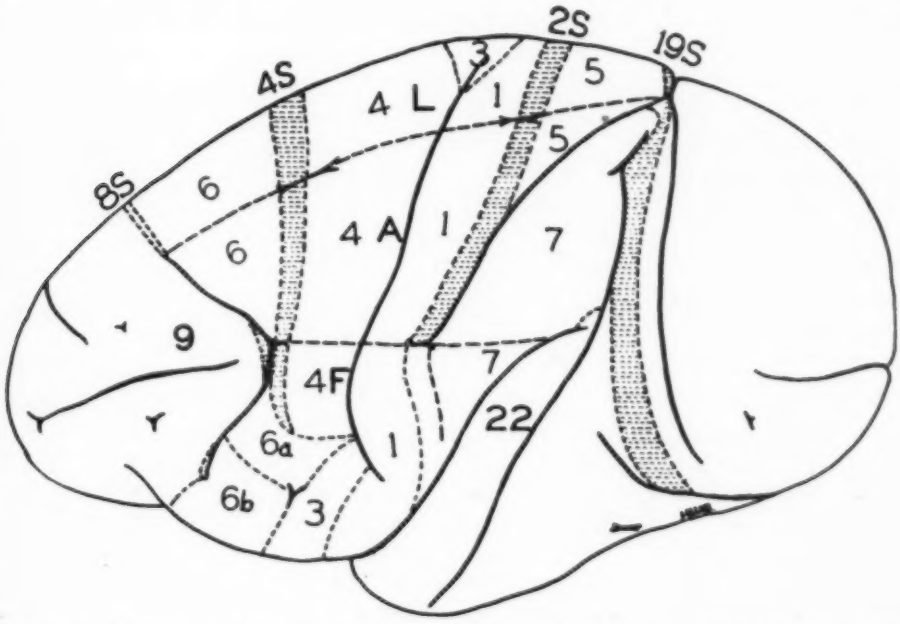

FIG. 1. *Macaca mulatta*. Diagram of sensory and adjacent cortex and convexity of hemisphere. Areas 6, 4s, 4, 3, 1, 2s, 5 and 7 constitute the sensory cortex. Letters L, A and F indicate the functional subdivisions for leg, arm and face respectively. = = = = = = = indicates areas from which suppression of electrical activity of the cortex can be obtained. Of these areas 8s and 19s are immediately adjacent to the sensory cortex but not part of it.

tion between 6bα and 6bβ. The subdivisions for leg, arm and face are indicated by the appropriate letters.

Since this work was completed a new finding of interest with respect to the sensory cortex has been obtained in experiments on the occipital lobe in which connection it will be fully reported later. In area A7 immediately below the sulcus interparietalis and sometimes confined to it lies a narrow strip of cortex which is fired from the occipital lobe. To date it is the only part of the sensory cortex fired by strychninization of the cortex anywhere outside the sensory cortex.

Insofar as the results merely confirm previous findings they will not be reiterated except by schematic representation in the conclusion. All results to be considered are of one of two types, namely the occurrence of the large

sudden voltages, called strychnine spikes from their appearance in the record, or the transient diminution or disappearance of electrical activity produced by strychninization of restricted areas and called the suppression of electrical activity. (i) Local strychninization of area 9 neither produces strychnine spikes (*i.e.*, "fires") nor reduces the electrical activity of (*i.e.*, suppresses) any area of the sensory cortex. Occasionally its strychninization is followed by increase of activity of several cortical areas but this occurs so irregularly and its site is so various that it must depend upon other variables than those controlled in these experiments. Except for this, the only effect of strychninization of area 9 is the firing of area 9. (ii) Local strychninization of area 8s fires area 8s locally and suppresses the electrical activity of the entire hemi-

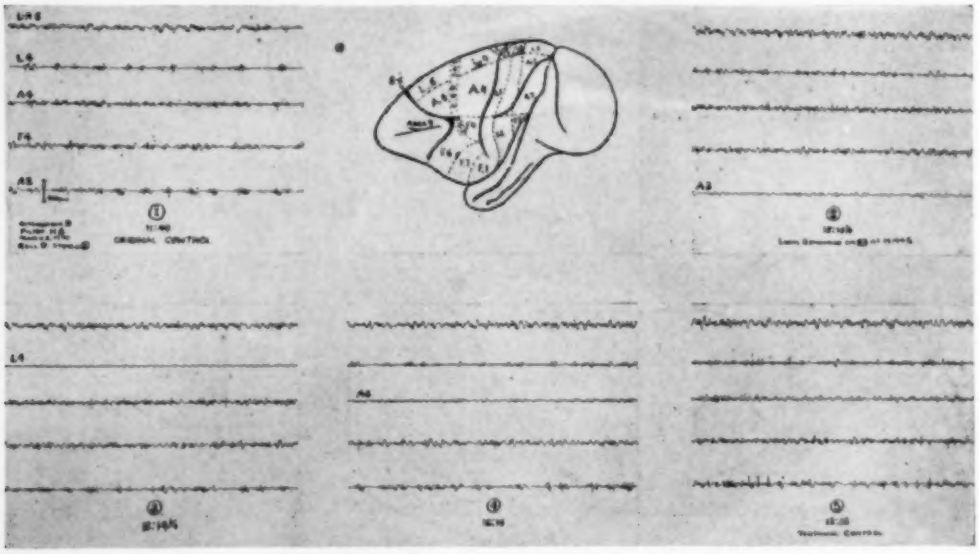

FIG. 2. March 2, 1940. *Macaca mulatta*. Dial. Strychninization of 8s showing suppression first seen in A2 later in L4 then in A4.

sphere.[5,12] The suppression appears first in regions nearest to area 8 and sweeps across the cortex. It usually begins 7 to 12 min. after the application of strychnine, lasts several minutes and requires about 15 to 20 min. to cross the sensory cortex. Figure 2 shows the records of such a suppression. (iii) Nothing new has been discovered with respect to strychninization of area 6 except that its anterior margin has several times been found well forward in the concavity of the sulcus arcuatus. (iv) Strychninization of 4s (the "strip" of Marion Hines[13,14]) has twice been accomplished without impinging on area 6 or area 4. (This was done only after experiments on the chimpanzee had shown the simplified picture which one might hope from a pure 4s strychninization.) It fired itself locally and *no other area* and it produced a suppression like that obtained from area 8. (v) Local strychninization of area 4 so far has yielded no new findings although on the basis of the finding on

the chimpanzee[6] one would have expected them. (vi) Local strychninization of area 3 fires itself very strongly and also areas 1, 2 and 5 but weakly. It produces very questionable firing, if any, in area 4 and perhaps (on one occasion) in area 7. (vii) Local strychninization of area 1 fires itself and areas 2, 5 and 7 strongly. It also fires weakly area 4 and possibly area 4s. (viii) Local strychninization of area 2s in the leg- or arm-subdivision fires itself only locally and gives a suppression of the entire cortex like that seen after strychninization of 8s and 4s. In the face area the attempt to strychninize area 2s has usually been unsuccessful and when successful has produced at most a weak suppression. (ix) No new findings have appeared after strychninization of area 5. (x) The same may be said of strychninization of area 7. (xi) Between area 7 and the first temporal sulcus there is sometimes, but not always, demonstrable a narrow triangular area—area 22—local strychninization of which fails to fire or suppress any part of the sensory cortex. (xii) Immediately posterior to the sensory cortex medial to the sulcus interparietalis and either separated from it only by the medial prolongation of area 22 or only by the first temporal sulcus lies area 19s strychninization of which produces a suppression like that obtained from 8s, 4s and 2s.

Two observations concerning the variations of the areas with respect to anatomical landmarks deserve mention.

(i) Area 8s, identified by the elicitation of eye movements and by strychninizations which yield suppression of electrical activity of the cortex, extends from the medial aspect of the hemisphere[12,15] laterally between areas L6 and 9 to almost the superior end of the medial limb of the sulcus arcuatus. From this point downward it usually passes into the anterior lip of the sulcus to emerge again only for a short distance as it crosses the concavity of the sulcus. However, it has appeared in one animal on the medial lip of the upper ramus, disappeared into the depths to reappear on the surface posterior and lateral to the sulcus in what is normally F6b; whereas, in another animal it crossed the concavity ending anterior to the tip of the inferior ramus.

(ii) Even greater variation occurs at the posterior margin of the sensory cortex above the sulcus interparietalis, where exploration is rendered difficult by a complex of veins in the vicinity of the sulcus parieto-occipitalis. Here precise localization can be obtained in only a small percentage of hemispheres, yet even these few disclose that area 5 sometimes extends into the sulcus parieto-occipitalis, and area 19 appears on the occipital lobe posterior thereto. In extreme cases area 5 stops short and area 19 appears in the parietal cortex and may even be separated from the sulcus parieto-occipitalis by an area a millimeter or two wide exhibiting properties of an area normally found in the occipital lobe. This latter arrangement resembles closely the relation to landmarks most frequently encountered in the chimpanzee.

Discussion

Two aspects of the present findings should be mentioned. The first of these is the existence of the suppressor bands 8s and 19s which bound the

sensory cortex, except for area 22 occasionally coming to the surface in front of the first temporal sulcus. As the animals used in this experiment were frequently used also for studying cortico-striatal projections,[5,8] it is possible to be certain that area 8s, like area 4s, projects to the nucleus caudatus. As it has already been shown that this projection is responsible for suppression from 4s, it seems only reasonable to conclude that it is likewise effective in the case of 8s, particularly in view of the finding that strychninization of the nucleus caudatus can itself produce suppression by blocking thalamo-cortical reverberations. As yet it has not been possible to demonstrate similar projections from areas 2s and 19s.

Since this statement was made Drs. Hugh Garol and Percival Bailey, working in this laboratory, have shown that strychninization of 2s does fire far posteriorly into the horizontal portion of the nucleus caudatus. This finding makes it advisable to expect connections of 19s to still more caudal portions of the nucleus caudatus.

There are, therefore, on the convexity of the hemisphere of the monkey four suppressor bands, two within and two just without the sensory cortex and all four are alike in their failure to fire other portions of the sensory cortex. Thus it resembles the chimpanzee's functional organization.

The second point requiring comment concerns the clarification of the findings in strychninization of 4s and 2s. In the case of 4s, the pure picture differs from the previous mixed (which we had mistaken for pure) merely by the omission of firing elsewhere. In the case of 2s (which we, lacking histological control, had tentatively called 1) the change is more complicated. Reference to the previous diagram of functional organization[7] shows that areas 2 and 5 had no difference as to other regions fired from them, a finding out of line with those for all other areas strychninized. The difference which now appears is that the area we had considered to be 5 failed to fire other parts of 5, whereas 2 did so. It is now clear that both were really area 5, and that within 5 the firing *is* more restricted in the most posterior portion. In the paper mentioned above all mention of leg or arm 3 was omitted, for it is difficult to strychninize it without touching area 1 and the difference in firing of other areas from 3 and 1 is not clear enough to be absolutely certain of the results. The same may be said of pickup electrodes intended for area 3, *i.e.*, disturbances from 1 may be included. It seems therefore wisest to omit area 3 from the schematic representation of the findings except for the face region. When these modifications are made the new scheme and the new map bear a still more striking resemblance to that of the chimpanzee, at least so far as the leg and arm regions are concerned.

As already stated, the work on the chimpanzee gave clues as to what might be expected in the monkey, for, being larger, the bands of the chimpanzee's cortex were more easily strychninized separately. Lest the work on the chimpanzee be thought to have been unduly influential in producing the similarities noted above it should be emphasized that, in the monkey, repeated careful attempts to distinguish two bands in area 4 and any subdivision into bands of area 5 or 7 have failed completely, except for the more

restricted firing in the posterior part of area 5. Whether the failure to find these differentiations is due to their non-existence or to the small size of the area to be subdivided cannot be determined by the procedures here employed. On the other hand, if one considers the threshold to electrical stimu-

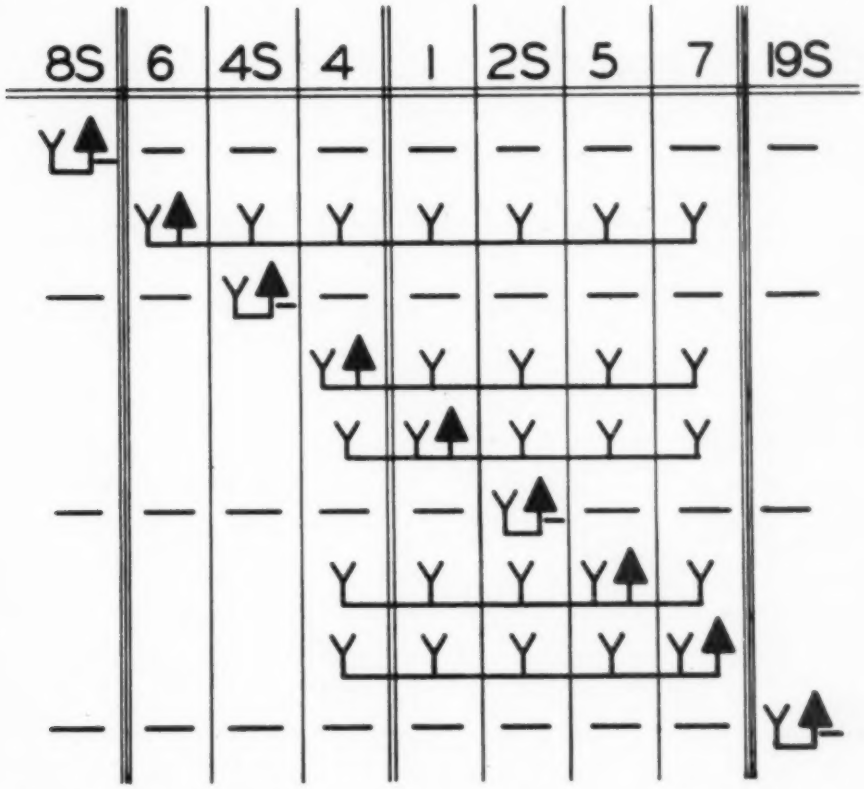

FIG. 3. Functional organization of sensory and adjacent cortex of leg- and arm-subdivisions. Solid triangle indicates location of strychnine; Y, the appearance of strychnine spikes; —, the suppression of electrical activity. Diagram indicates the maximum results obtained by strychninizations confined to the areas indicated. Double lines indicate sulcus centralis; triple lines, the confines of the sensory cortex.

lation, area 4 of the monkey is divisible into two bands, and, as excitation by strychninization remains generally more nearly confined to its site than does that by electrical stimulation, one would expect that a difference of functional organization of these bands in 4 would certainly be discoverable it if exists. It was hoped that the somewhat larger brain of a large mangabey might have sufficiently larger areas to show separate bands in areas 5 and 7 but it failed so to do. In this manner and in these respects the monkey's brain seems to lack differentiations found in the chimpanzee and here the maps remain significantly dissimilar.

SUMMARY

The accompanying schematization (Fig. 3) indicates only those findings

which are well established in the monkey and summarizes the results of the present and previous investigations of the functional organization of the sensory and adjacent cortex.

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